VOL. LXXVI JANUARY, 1930 NEW SERIES VOL. 29, NO. 1

JOURNAL
of the
American Veterinary Medical Association

FORMERLY
AMERICAN VETERINARY REVIEW
(Original Official Organ U. S. Vet. Med. Ass'n.)
EDITED AND PUBLISHED FOR
The American Veterinary Medical Association

CONTENTS

Communications relating to publication, subscriptions, advertisements and remittances for the JOURNAL OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION, as well as matters pertaining to the American Veterinary Medical Association and membership, should be sent to Dr. H. Preston Hoskins, Secretary-Editor, 716 Book Bldg., Detroit, Mich.

$4.00 per annum Foreign $5.00; Canada $4.35 Single Copies 40 cts. in U. S.

Entered as Second Class Matter, March 15, 1923, at the Post Office at Detroit, Mich., under Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917; authorized October 26, 1918.

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Vol. LXXVI, N. S. Vol. 29 January, 1930 No. 1

VOLUME SEVENTY-SIX

How many of those who look at the date-line of this issue will stop to realize just what information lies hidden therein. Volume seventy-six! Over fifty years of continuous publication—almost fifty-three, to be more nearly correct; thirty-eight as the *American Veterinary Review* (1877-1915) and fifteen as the Journal of the A. V. M. A. (1915-1930). During this period there have been 636 numbers issued. We will not calculate the number of pages —it would be too much of a task. Suffice it to say, however, that a complete set of the *Review* and Journal, bound and placed on a bookshelf, requires about thirteen running feet. On another page of this number will be found the minutes of the special meeting of the Executive Board, held in Chicago, the past month. Several very importnat matters in connection with the Journal were considered at that meeting. One was to increase the size of the Journal, in order to be able to accommodate the increasing amount of material which we are called upon to publish. The shortness of time available prevented us from enlarging the size of this issue, but the February issue will show the increase. This number will contain about a dozen of the papers read at the 1929 convention in Detroit. In March we will present the complete proceedings of the 1929 meeting of the United States Live Stock Sanitary Association, which means an issue of about 300 pages.

AMERICAN MEDICAL ASSOCIATION ACTS

At its recent meeting, the Board of Trustees of the American Medical Association voted to increase ths subscription price of the JOURNAL of the A. M. A., including fellowship dues, from $5.00 to $7.00 per year. The action was taken in accordance with authorization by the House of Delegates at the annual meeting held in Portland, the past year.

According to a statement sent all subscribers, the advisability of the increase should be apparent to all concerned. The expansion of the work of the Association and particularly the extension of service rendered fellows and subscribers during the last years is widely recognized. New undertakings have proved to be heavy drains on the finances of the Association. Special committees have made grants for scientific research in different fields for the councils on medical education, on pharmacy and chemistry, on physical therapy, and on scientific assembly and these bodies are functioning for the good of medicine and for the public health without asking any financial return from the medical profession or the public. The statement goes on to enumerate other services which are being rendered by the Association, all of these being well known to those who have followed carefully the annual reports made by the Board of Trustees.

The present plans of the Board of Trustees contemplate, among other things, new buildings, a national scientific exhibit, extension of library and bibliographic service and a wider extension of the help that the Association can render to the individual practitioner.

It would appear that the American Medical Association finds itself in substantially the same situation in which the American Veterinary Medical Association finds itself right now. For a number of years the A. V. M. A. has gone right on expanding its field of activities and increasing the services rendered members without any compensating increase of income. The limit has been reached and, if there is to be no curtailment of our activities and no let-up in the work already undertaken, some action similar to that just taken by the American Medical Association appears to be imperative.

Dues Are Due

Notices for the payment of the 1930 dues were mailed to our members late in November. During December about 35 per cent of our membership sent in their dues. Did you?

APPLICATIONS FOR MEMBERSHIP

During the year 1929, there were 633 applications for membership listed in the JOURNAL—a very satisfactory showing; in fact an increase of 171 over the year 1928 and more than double the number for 1927. We would like to make 1930 just as good a year as the one just closed and some hard work will make it possible. In addition to bettering the 1929 figure, there is another goal to attain—the 5000-member mark. With approximately 4500 members on the books at the turn of the year, and with a normal loss of 100 members for various reasons during 1930, a gain of approximately 600 new members this year will give us the desired membership strength of 5000 by the end of 1930—a goal hitherto not attained. Let us repeat: Some hard work will do it.

Do not forget the plan under which applications for membership are now being handled. Here is a quotation from the By-laws:

> Applications for membership shall be made upon blanks furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Applications must be accompanied by a membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Applications must be filed with the Secretary and examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of the applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

ABREVAYA, LEON — c/o A. S. P. C. A., 24th St. & Ave. A, New York, N. Y.
D. V. M., Cornell University, 1929
Vouchers: L. D. Mersch and C. E. Hayden.

BROWN, JOHN C. — Joy, Ill.
D. V. S., University Veterinary College, 1904
Vouchers: W. H. Welch and C. C. Hastings.

CALDWELL, GEO. H. — 1904 W. North Ave., Chicago, Ill.
D. V. M., Chicago Veterinary College, 1915
Vouchers: L. A. Merillat and J. F. DeVine.

EILER, EDWARD JOHNSON — Mt. Sterling, Ill.
M. D. V., McKillip Veterinary College, 1910
Vouchers: W. B. Holmes and C. C. Hastings.

GATES, ORVAL L. c/o A. S. P. C. A., 24th St. and Ave. A, New York, N. Y.
D. V. M., Colorado Agricultural College, 1925
Vouchers: L. D. Mersch and Geo. H. Glover.

HAWORTH, CHARLES C. Donnellson, Ill.
M. D. C., Chicago Veterinary College, 1910
Vouchers: C. C. Hastings and W. H. Welch.

HERRON, HUGH Watseka, Ill.
D. V. M., Indiana Veterinary College, 1913
Vouchers: C. C. Hastings and C. E. Lucas.

HOWE, EDWARD C. Troublesome, Colo.
D. V. M., Colorado Agricultural College, 1929
Vouchers: Geo. H. Glover and I. E. Newsom.

KILFOY, LEO T. 2508 Lawrence Ave., Chicago, Ill.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: Bernard F. Humphreys and Geo. P. Frost.

KINDRED, T. H. Sioux Falls, S. Dak.
D. V. M., McKillip Veterinary College, 1917
Vouchers: N. L. Nelson and G. P. McCue.

LAWTON, WILLIAM CURTIS 524 N. Jefferson St., Madison, Ind.
D. V. M., Indiana Veterinary College, 1918
Vouchers: R. R. Newman and Frank H. Brown.

OSTEEN, ANDREW JACKSON Belhaven, N. C.
D. V. M., Georgia State College of Agriculture, 1928
Vouchers: Wm. Moore and W. C. Dendinger.

PIRIE, LESLIE DANIEL Riverdale, Calif.
D. V. M., San Francisco Veterinary College, 1914
Vouchers: Joseph M. Arburua and John McInnes.

Applications Pending

SECOND LISTING

Ashmore, C. Delmer, 209 Livestock Exchange, Union Stock Yds., Los Angeles, Calif.
Bett, Thomas Pattullo, 12041 E. Jefferson Ave., Detroit, Mich.
Cobbett, Norman G., 521 N. Cherry St., Galesburg, Ill.
Houser, Roy, Bourbon, Ind.
Kiff, Walter J., 328½ Franklin St., Michigan City, Ind.
Magrane, Harry J., 1207 Lincoln Way, West, Mishawaka, Ind.
Mathews, Frank P., 204 S. Salisbury, West Lafayette, Ind.
Striggle, Claude Walter, South Whitley, Ind.
Warren, Dayton McRae, 529 N. Rodney St., Helena, Mont.

The amount which shall accompany an application filed this month is $10.00, which covers membership fee and dues to January 1, 1931, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 6-10, 1930. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

Kansas State Veterinary Medical Association and Kansas State Agricultural College Conference for Veterinarians. Kansas State Agricultural College, Manhattan, Kans. January 7-8, 1930. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kans.

University of Pennsylvania, Conference of Veterinarians at. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 8-9, 1930. Dr. Louis A. Klein, Dean, 39th St. & Woodland Ave., Philadelphia, Pa.

Ohio State Veterinary Medical Association. Deshler-Wallick Hotel, Columbus, Ohio. January 8-9, 1930. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Oklahoma State Veterinary Medical Association. Huckins Hotel, Oklahoma City, Okla. January 13-14, 1930. Dr. C. H. Fauks, Secretary, 1919 W. Ash St., Oklahoma City, Okla.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. January 14, 1930. Dr. J. B. Jaffray, Secretary, 2956 Washington Blvd., Chicago, Ill.

Kansas City Association of Veterinarians. New Baltimore Hotel, Kansas City, Mo. January 14, 1930. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.

Tennessee Veterinary Medical Association. Knoxville, Tenn. January 14-15, 1930. Dr. A. C. Topmiller, Secretary, Box 238, Murfreesboro, Tenn.

Intermountain Livestock Sanitary Association. Ogden, Utah. January 14-15, 1930. Dr. Cecil Elder, Secretary, University of Wyoming, Laramie, Wyo.

Wisconsin Veterinary Medical Association. Madison, Wis. January 14-16, 1930. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Iowa Veterinary Medical Association. Ft. Des Moines Hotel, Des Moines, Iowa. January 14-17, 1930. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. January 15, 1930. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles.

Maryland State Veterinary Medical Association. Medical Hall, 1211 Cathedral St., Baltimore, Md. January 16, 1930. Dr. E. M. Pickens, Secretary, College Park, Md.

Texas, State Veterinary Medical Association of. Raleigh Hotel, Waco, Texas. January 16-17, 1930. Dr. D. Pearce, Secretary, Box 335, Leonard, Texas.

Minnesota State Veterinary Medical Association. Hotel Lowry, St. Paul, Minn. January 16-17, 1930. Dr. C. P. Fitch, Secretary, University Farm, St. Paul, Minn.

Cornell University, Annual Conference for Veterinarians at. Cornell University, Ithaca, N. Y. January 16-17, 1930. Dr. P. A. Fish, Dean, New York State Veterinary College, Ithaca, N. Y.

South Dakota Veterinary Medical Association. Hotel Cateract, Sioux Falls, S. Dak. January 16-17, 1930. Dr. W. P. Brower, Secretary, Canton, S. Dak.

Indiana Veterinary Medical Association. Hotel Severin, Indianapolis, Ind. January 21-23, 1930. Dr. R. H. Boyd, Secretary, 1422 N. Capitol Avenue., Indianapolis, Ind.

Missouri, University of, Special Short Course for Veterinarians. University of Missouri, Columbia, Mo. January 21-24, 1930. Dr. J. W. Connaway, University of Missouri, Columbia, Mo.

Keystone Veterinary Medical Association. Philadelphia, Pa. January 22, 1930. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

Nevada State Veterinary Association. Reno, Nevada. January 22, 1930. Dr. Edward Records, Secretary, University of Nevada, Reno, Nevada.

Virginia Polytechnic Institute Veterinary Short Course. Blacksburg, Va. January 27-31, 1930. Dr. Russell A. Runnells, Virginia Polytechnic Institute, Blacksburg, Va.

South Carolina Association of Veterinarians. Columbia, S. C. January 28, 1930. Dr. M. R. Blackstock, Secretary, 157 Hampton Ave., Spartanburg, S. C.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 28-31, 1930. Dr. Ward Giltner, Dean, Division of Veterinary Science, Michigan State College, East Lansing, Mich.

Delaware Veterinary Medical Association. Dover, Del. January 29, 1930. Dr. J. R. Porteus, Secretary, Box 365, Smyrna, Del.

Alabama Veterinary Medical Association and Short Course for Practitioners. Auburn, Ala. February 2-7, 1930. Dr. C. A. Cary, Secretary, Alabama Polytechnic Institute, Auburn, Ala.

Illinois Veterinary Conference, University of. Urbana, Ill., February 10-12, 1930. Dr. Robert Graham, University of Illinois, Urbana, Ill.

Southeastern States Veterinary Medical Association. New Orleans, La. February 10-11, 1930. Dr. M. R. Blackstock, Secretary, 157 Hampton Ave., Spartanburg, S. C.

THE PATHOGENICITY OF ABORTION VACCINES FOR GUINEA PIGS*

By J. P. TORREY, *Department of Bacteriology, and*
E. T. HALLMAN, *Department of Animal Pathology*
Michigan State College, East Lansing, Mich.

In a paper on the abortion problem published in the January, 1929, issue of *The North American Veterinarian*, Dr. E. A. Cahill makes the following statements:

> I have excellent reason to believe that every reputable producer of vaccine in this country has taken the necessary steps to make certain that the organisms in abortion vaccine while highly antigenic are non-virulent. There is unmistakable evidence of the fact that undulant fever in the human is traceable to cows and other animals which are infected with abortion disease. If abortion vaccine contained virulent organisms capable of reproducing the disease there might be serious objections to its use on account of its possible transmission to the human but since, as stated above, the organisms in abortion vaccine are non-virulent there is no danger on this score.

Because of the tremendous importance of the matter of virulence of organisms used as living vaccines and the wide publicity given to the above statements, the authors decided to test a number of widely advertised abortion vaccines for virulence. Since there are obvious difficulties in determining the virulence of *Brucella abortus* for cattle and since *Br. abortus* produces a characteristic disease in guinea pigs, the guinea pig was used as the test animal in this work.

According to a report of the Division of Virus-Serum Control, United States Bureau of Animal Industry, issued March 31, 1928, there are twenty biological firms which have licenses to produce bovine abortus vaccine. Since that time a few others have been added to the list. As it was a rather large undertaking to test all of the vaccines on the market, it was decided to select nine presumably reputable and well-known manufacturers located geographically so as to represent the United States east of the Rockies.

In February, 1929, one dose of vaccine was purchased from each of these nine manufacturers and subjected to viability and virulence tests. For convenience we will number these vaccines from one to nine inclusive. After the guinea pigs used for virulence tests had been injected with the vaccine, 0.1 cc was

*Presented at the sixty-sixth annual meeting of the American Veterinary Medical Association, Detroit, Mich., August 13-16, 1929. Published with the permission of the Director of the Agricultural Experiment Station, as Journal Article No. 17, N. S.

placed on each of two plain liver-agar plates, two liver-agar plates containing carbol-fuchsin, and two liver-agar plates containing thionin. The vaccine was then spread over the plates by the use of sterile glass rods. The plates were incubated at 37°C. for five days, then placed in ten per cent carbon dioxid and incubated three days. All of the cultures reached their maximum growth aerobically. Transfers were made to agar slants and the agglutinability of the organisms from each living vaccine was tested with known positive bovine serum. The data relative to viability and agglutinability are recorded in table I.

Vaccines 3, 4 and 6 gave no growth and vaccine 5 gave only slight growth on carbol-fuchsin agar. The remaining five gave good growth on all plates.

TABLE I—*Viability and agglutinability of organisms in nine commercial abortion vaccines*

Vaccine	Expiration Date on Label	Received (1929)	Culture Made (1929)	Growth on Culture Media			Agglutinability of the Vaccine Organism
				Plain	Carbol-fuchsin	Thionin	
1	7-10-29	2-15	2-20	+++	+++	+	1-3200
2	1-16-30	2-15	2-20	++	+	—	1-3200
3	3-12-29	2-15	2-20	—	—	—	
4	5- 3-29	2-15	2-20	—	—	—	
5	5-30-29	2-15	2-20	—	+	—	1-1600
6	5- 7-29	2-15	2-20	—	—	—	
7	7-31-29	2-15	2-20	+++	+++	++	1-1600
8	9-18-29	2-15	2-20	+++	+++	+	1-1600
9	8-20-29	2-27	2-28	+++	+++	+	1-3200

— = no growth.
+ = 5 to 100 colonies per plate.
++ = numerous individual colonies well scattered over plate.
+++ = more or less diffuse growth covering plate.

For virulence tests, seventy-two guinea pigs (eight for each vaccine) were purchased from a breeder from whom we have been purchasing guinea pigs for experimental purposes for several years and who has maintained his reputation of always furnishing healthy and satisfactory pigs. These were delivered to a building on the campus in which no experiment or other animals had ever been kept. They were weighed and placed in sterilized cages, each cage containing four pigs, and during the entire tests they were fed and watered by one attendant who had no contacts with other animals of any kind. Eight pigs were used for each vaccine. The first and second pigs of each group received 2 cc of vaccine, the third and fourth pigs received 1 cc of vaccine, the fifth and sixth pigs received 0.5 cc of vaccine and the seventh and eighth

pigs received 0.25 cc of vaccine. The first four and the last four pigs of each group were placed in separate cages. All cages were separated by metal partitions so that there was no contact between pigs of different cages. The odd-numbered pigs received the vaccine intraperitoneally. The even-numbered pigs received the vaccine subcutaneously.

Six to eight weeks from the time of inoculation, all pigs were weighed and killed and blood was collected for the agglutination tests, the gross pathological lesions recorded, and material for histological examination was saved. Parts of the lung, liver, spleen, kidney and reproductive organs were smeared on plain

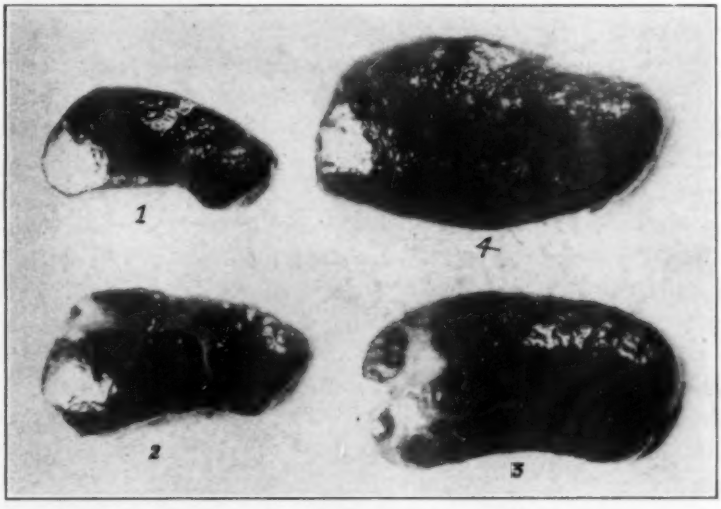

FIG. 1. Spleen 1 from 347-gram, normal guinea pig, for comparison with the remaining three diseased spleens (weight, 0.81 grams). Spleens 2, 3 and 4 from guinea pigs inoculated with *Br. abortus*, obtained from first group of pigs receiving injections of vaccine 1. (Spleen 2, 1.97 grams; spleen 3, 3.95 grams; spleen 4, 4.3 grams.)

liver-agar plates and on liver-agar plates containing gentian-violet. These plates were incubated at 37° C. for five days. At this time transfers were made to agar slants of colonies resembling *Br. abortus*, and later their agglutinability tested with bovine serum that was positive for *Br. abortus*.

DISCUSSION

Vaccine 1 contained a pure culture of living *Br. abortus*. The organisms were recovered from the spleens of pigs 2 and 3, and the lungs of pig 4. None of the pigs showed any distinct lesions. In pig 1, the spleen was recorded as suspicious and in pig 4, the lungs and kidney were recorded as suspicious. From these re-

sults it would appear that vaccine 1 consisted of abortion bacilli possessing little, if any, virulence. However, another group of three pigs was inoculated with the organism isolated from the spleen of pig 2, three pigs inoculated with the organism obtained from the spleen of pig 3, and three pigs inoculated with the organism obtained from the lung of pig 4. The pigs of each group of three received 1/20, 1/10 and 1/5, respectively, of a 24-hour agar slant culture of the organism. Of these nine pigs autopsied six weeks later, five show distinct and well-marked gross lesions on autopsy.* The blood serum from eight of these pigs reacted (1-500 or more) to the abortus organism, and one reacted at 1-25 only. *Br. abortus* was recovered from seven of the nine pigs injected.

The three enlarged spleens shown in figure 1 were from three of these five pigs. Histological examination of the organs of these pigs has not been completed at this writing.

It therefore appears that the organisms in vaccine 1, are not non-virulent but, while apparently very much attenuated, are capable of acquiring much virulence under favorable conditions.

Vaccine 2 was viable. The organism was not recovered from any of the pigs. The spleen of pig 9 showed slight microscopic foci recorded as positive; one lymph-gland of pig 10 was recorded as positive; the liver and kidney of pig 12 were recorded as suspicious. The kidney of pig 13 was recorded as suspicious and the spleen and one lymph-gland as positive; pig 14, suspicious in the liver and kidney; and pig 16, suspicious in the spleen. Since the abortion bacillus was not obtained from any of the organs of the pigs inoculated with this vaccine, we have not been able to subject it to the same tests for virulence to which vaccine 1 was subjected. However, with three pigs out of seven showing slight lesions suggestive of *Br. abortus* infection, and three of the remaining four showing suspicious lesions, we cannot feel that the organism is without virulence.

Investigations with this product are being continued and will be reported at a later date.

With vaccines 3, 4 and 6, we were unable to get any growth on cultures, although these cultures were made 20, 72 and 76 days, respectively, prior to the expiration date on labels.

The results of virulence tests of vaccine 4 are somewhat surprising and confusing. All pigs in this group reacted negatively

*Microscopic examination of the organs of these pigs reveals well-marked lesions in six, and slight lesions in two others.

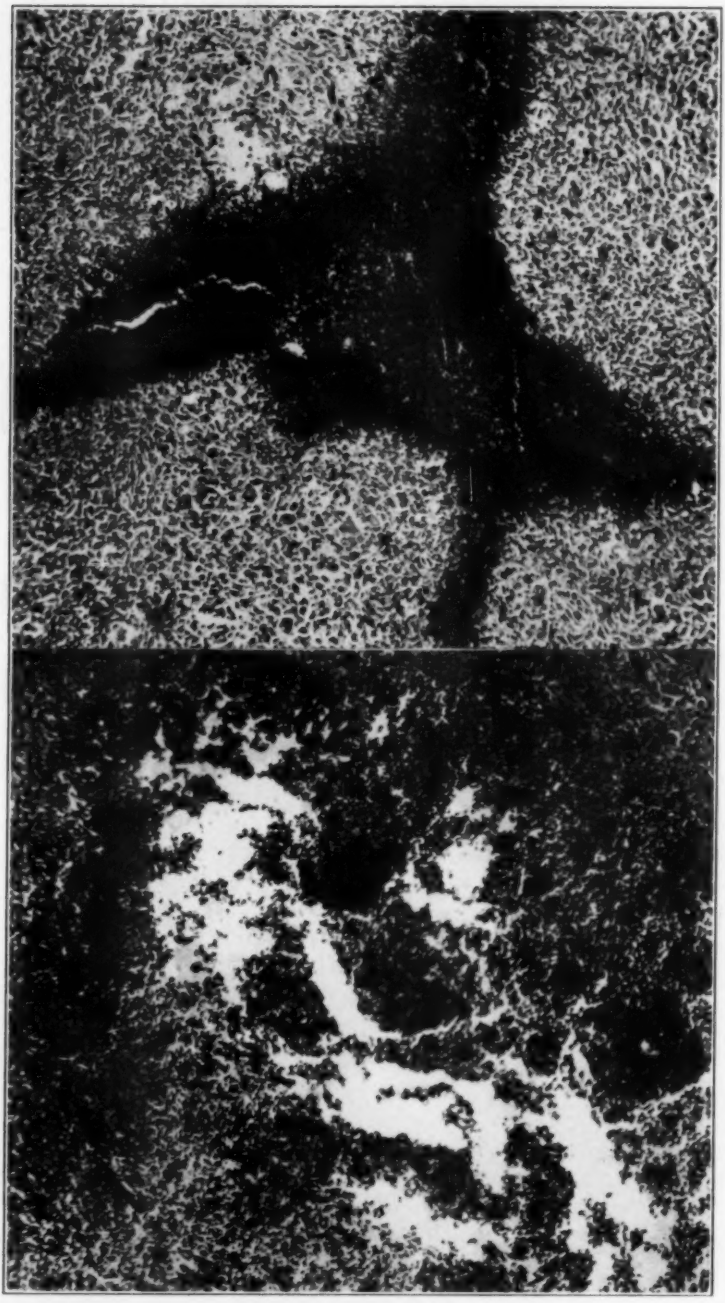

FIG. 2. (Above) Lymph-gland of pig 57 (vaccine 8). Extensive hyperplasia of reticulo-endothelial cells and necrosis. ca. x 100.
FIG. 3. (Below) Abscessed lymph-gland of pig 57 (vaccine 8). ca. x 100.

to the agglutination test at the time of autopsy except pigs 29 and 31, which were strongly positive. These pigs received the inoculations intraperitoneally and also received the smallest amounts of vaccine. *Br. abortus* was recovered from only one pig (29), in

FIG. 4. One of several abscesses in uterine mucosa of pig 72 (vaccine 9). *Br. abortus* was cultured from uterus. ca. x 100.

lung, liver, spleen and testicle. Only the lungs of this pig showed slight lesions. *Br. abortus* was not recovered from pig 31, although there were well marked lesions in the lungs, kidney, spleen, and

TABLE II—*Pathogenicity tests of organisms in abortion vaccines*

G. P.	Change in Weight on Experiment (Grams)*	Agglutination Reaction At Time of Autopsy†	Organs From Which Br. Abortus Was Recovered at Autopsy‡	Lesions Based on Gross and Microscopic Study§					
				Lung	Liver	Kidney	Reproductive Organs	Spleen	Lymph-glands
			Vaccine 1						
1	+19	++++P	None	—	—	—	—	Sl	—
2	—6	+++++	Sp	—	—	—		—	—
3	—34	++	Sp	—	—	—	—	—	—
4	+20	—	Lu	Sl	—	Sl	—	—	—
5	+90	++P	None	—	—	—	—	—	—
6	+45	+++++	None	—	—	—	—	—	—
7	+43	++P	None	—	—	—	—	—	—
8	+57	+P	None	—	—	—	—	—	—
			Vaccine 2						
9	+180	—	None	—	—	—	—	+	—
10	+68	—	None	—	—	—	—	—	1 gland +
11		Died soon after injection							
12	+153	—	None	—	Sl	Sl	—	—	—
13	+109	+++P	None	—	—	Sl	—	+	1 gland +
14	+127	++P	None	—	Sl	Sl	—	—	—
15	+112	+P	None	—	—	—	—	—	—
16	+65	++P	None	—	—	—	—	Sl	—
			Vaccine 3						
17 to 24	All records negative								

TABLE II—*Pathogenicity tests of organisms in abortion vaccines—Continued*

G. P.	CHANGE IN WEIGHT ON EXPERIMENT (GRAMS)*	AGGLUTINATION REACTION AT TIME OF AUTOPSY†	ORGANS FROM WHICH BR. ABORTUS WAS RECOVERED AT AUTOPSY‡	LESIONS BASED ON GROSS AND MICROSCOPIC STUDY§					
				LUNG	LIVER	KIDNEY	REPRODUCTIVE ORGANS	SPLEEN	LYMPH-GLANDS
			Vaccine 4						
25	+108	—	None	—	—	—	—	—	—
26	+221	—	None	—	—	—	—	—	—
27	+129	—	None	—	—	—	—	—	—
28	+82	—	None	—	—	—	—	—	—
29	+24	++++P	Sp, T, Lu, Li	+	—	—	—	—	—
30		—	None	—	—	—	—	—	—
31	+85	+++++	None	++	+	++	Sl	++	++
32	+43	—	None	—	—	—	—	—	—
			Vaccine 5						
33	+44	—	None	—	—	—	—	Sl	1 gland ++, 1 gland +
34	+46	++P	None	—	—	—	—	—	1 gland ++
35	+130	++P	None	—	Sl	Sl	—	—	—
36	+15	+++	None	—	—	—	—	Sl	—
37	+23	—	None	—	—	—	—	—	—
38	—32	—	None	—	—	—	—	—	—
39	+12	—	None	—	—	—	—	—	—
40	+110	+	None	—	Sl	—	—	—	—
			Vaccine 6						
41 to 48	All records negative								

TABLE II—*Pathogenicity tests of organisms in abortion vaccines—Continued*

G. P.	Change in Weight on Experiment (Grams)*	Agglutination Reaction At Time of Autopsy†	Organs From Which Br. Abortus Was Recovered at Autopsy‡	Lesions Based on Gross and Microscopic Study§					
				Lung	Liver	Kidney	Reproductive Organs	Spleen	Lymph-glands
Vaccine 7									
49	—2	+++++	Lu, Li, K, T, Sp	+	+++	+		++	++
50	+73	+++++	K, Sp	—	+	—	+	+	+++
51	+85	+++++	K, Sp	—	+	+	++	++	++
52	+40	+++++	Sp, K, T, Li	++	+	Sl	—	Sl	++
53	—102	+++++	Lu, T, Li, Sp, K	++	+	Sl	—	—	—
54	+204	+++++	Li, Sp, T	—		—	—	++	++
55	+73	+++++	Li, Sp, T	—	+	+	+	+	++
56	+51	+++++	Li, Sp, K	—	+	—	—	++	++
Vaccine 8									
57	—14	+++++	Lu, Li, K, Sp	+	++	+	F, +	+++	++++
58	+72	+++++	Sp, Li, K	++	+	—		+	++
59	+43	+++++	Li, Sp, K	++	+	+	F	++	++
60	+76	+++++	Sp, K	++	+	+	F, —	++	++
61	—18	+++++	Li, Sp, K	—	++	—	—	+++	+++
62	+35	+++++	K, Sp	—	+	+	F, —	+	++
63	+95	+++++	Li, Sp, K	—	+	—	F, —	+	+
64	+41	+++++	Li, Sp, K, Lu	++	+++	+	F	+++	++
Vaccine 9									
65	+82	+++++	Li, Sp, T, Lu, K	+	+	Sl	++++	++	++
66	+40	+++++	Li, Sp, K	—	+	—	F	+++	+++
67	+140	+++++	Lu, Sp	++	—	—	F	+++	+++
68	+55	+++++	Li, Sp, K	++	+	—	F	++	+++
69	+144	++++	Lu, Sp	+	+	—	F, —	++	++
70	+77	+++++	Lu, Li, Sp, K	++	+++	+	F	+++	++
71	+97	+++++	Sp	—	+	+		+	+
72	+139	+++++	U, Sp, K	—	Sl	—	F, ++++	+++	++

* + = grams gained in weight. — = grams lost in weight.
† + =1-25. ++ = 1-50. +++ = 1-100. ++++ = 1-200. +++++ = 1-500.
‡ K = kidney. Li = liver. Lu = lung. Sp = spleen. T = testes. U = uterus.
§ — = negative. + = slight. Few microscopic focal lesions in section. ++ = well marked. Fairly numerous microscopic or macroscopic lesions or both. +++ = well marked. Necrosis without abscessation. ++++ = well marked. Necrosis and abscessation. Sl = one or few atypical microscopic lesions. F = female.

lymph-glands; slight lesions in the liver, and suspicious lesions in the testicles, suggestive of *Br. abortus* infection. While we have no satisfactory explanation for these results, we venture the hypotheses that pigs 29 and 31 received clumps of organism, some of which were viable, although the cultural tests of the vaccine indicate that the organisms were dead.

With vaccine 5, the results of the viability tests are somewhat confusing. No growth was obtained except a few colonies in plates of liver-agar containing carbol-fuchsin. We are inclined to think that these results indicate that all but a few of the organisms in the vaccine were dead. The agglutination reactions of pigs 34, 35 and 36 seem to strengthen this opinion. Pig 33 showed well-marked lesions in one lymph-gland, slight lesions in another gland and suspicious lesions in the spleen. Pig 34 showed well-marked lesions in only one gland. Pig 35 showed suspicious lesions in the liver and kidney; pig 36, suspicious in the spleen; and pig 40, suspicious in the liver.

Vaccines 7, 8 and 9 consist of living and highly virulent strains of *Br. abortus* for guinea pigs. These strains show much greater virulence for guinea pigs than many of the recently isolated bovine strains with which it has been our privilege to work during the past few years.

SUMMARY

Abortion vaccines from nine manufacturers were studied for viability and virulence. Of these our cultural data indicate that three were non-viable although tested 20, 72 and 76 days, respectively, before the expiration date stamped on labels. The fourth failed to grow except for a few colonies on carbol-fuchsin agar, indicating that the vaccine contained a few viable organisms. The virulence tests with two of these four indicate that they contained a few viable, virulent organisms. The five remaining vaccines were viable. Two of these appear to be highly attenuated but not non-virulent. One of the two apparently attenuated vaccines has been shown to consist of organisms possessing the property of acquiring considerable virulence when subjected to favorable conditions for the organisms. Three vaccines showed more virulence for guinea pigs than many recently isolated strains from aborting cattle.

In checking up on the number of organisms per dose recommended by the manufacturers, we note that the number of organisms per dose varies from 200 billion, in one of the most highly attenuated vaccines, to 1200 billion of one of the most virulent vaccines.

PRELIMINARY REPORT ON THE RELATION OF BACT. ABORTUS BANG TO FISTULAE, POLL-EVIL AND OTHER SUPPURATIONS OF HORSES*

By C. P. Fitch, A. L. Delez *and* W. L. Boyd

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The importance of fistulae and poll-evil in horses is still very great. The incapacity caused by these diseases in this group of animals is so large that all veterinarians, especially country practitioners, recognize poll-evil and fistulae as important economic factors. Many things have been described as the etiological agents. Trauma and pyogenic bacteria have been most commonly implicated. Clinicians have recognized different forms or types of the disease and some veterinarians have ascribed different etiological agents. Williams,[1] in 1908, divided fistulous withers into four classes: (1) superficial fistulous withers, referable to a direct traumatism with primary cutaneous lesions (false fistulous withers), (2) lymphoma of the withers, (3) abscess of the withers, and (4) fistula of the withers resulting from (2) and (3). In general, the same groups would apply to poll-evil. Williams further states:

> Class 1, originating from an evident external traumatism, is comparatively rare, there having been three cases among the 102 cases in our clinic, or 3 per cent, while in the 43 cases of poll-evil, none showed evidence of external injury. . . . . The malady is rural rather than urban. Were traumatisms the cause, the reverse should be true.

Davis,[2] an English veterinarian, does not recall an instance, in a long experience, where poll-evil or fistulae could be traced to injury. He observes that horses used continuously in cities are seldom affected with these diseases. "I should say that a hundred agricultural horses are affected for every one used constantly in town work, and *never turned out to grass.*" Dr. Davis believes that the condition is infectious, as glanders or tuberculosis.

Various pyogenic organisms have been isolated many times from poll-evils and fistulae. Gay[3] found three varieties of organisms in seven cases of fistulous withers. These were a streptococcus and a yellow and white micrococcus. In two cases of poll-evil, a streptococcus and a white micrococcus were cultured respectively.

*Presented at the sixty-sixth annual meeting of the American Veterinary Medical Association, Detroit, Mich., August 13-16, 1929. Published with the approval of the Director, as Paper 878 of the Journal Series of the Minnesota Agricultural Experiment Station.

TABLE I—*Pyogenic localization of Bang infection in horses (Rinjard and Hilger)*

No.	Sex	Date of Examination	Site of Localized Lesions	Sanitary Condition of Cattle on the Premises	Agglutination Titer	Results of Pus Examinations: Smears, Cultures, Guinea Pig Inoculations
1H	Mare	12–30–22	Withers	Infected	+ 1–200 Partial 1–400	Chronic infection. Mixed bacterial flora
2H	Mare	12–30–22	Withers and poll. Suppuration since Sept. Necrosis of spinous processes	Infected	+ 1–400	Chronic infection. Mixed bacterial flora
3H	Gelding	2–17–23	Withers	Infected	+ 1–100	Open lesion. Mixed bacterial flora. Not examined
4G	Mare	3–14–23	Withers	Infected	+ 1–100	Not examined
5H	Mare	9–18–23	Withers	Non-infected	0	Not suspected
6H	Mare	12–4–23	Poll	Infected	+ 1–500 Partial 1–1000	Suspect smears. Cultures sterile
7H	Mare	1–2–24	Withers	Infected	+ 1–1000	Suspect smears. Cultures sterile
8H	Mare	1–14–24	Poll	Infected	+ 1–300	Recurrent infection. Mixed bacterial flora
9H	Mare	2–3–24	Poll	Not under control. Bang infection exists in the neighborhood	+ 1–300	Stained smears suspected. Cultures contaminated
10H	Mare	6–12–15	Withers	Infected	+ 1–100	Smears suspected. Cultures sterile
11H	Mare	3–24–27	Withers	Infected	+ 1–100	Not examined
12H	Mare	5–24–27	Withers	Non-infected	0	Suspect smears. Cultures sterile. Inoculation negative
13H	Mare	6–13–27	Withers	Infected	+ 1–300	Suspect smears. Cultures sterile. Inculations positive (4 passages). Followed by isolation of bacillus of Bang
14H	Mare	8–25–27 9–2–27	Withers	Infected	+ 1–100 Partial 1–300	Suspect smears. Cultures sterile Inoculations positive (2 passages). Followed by isolation of bacillus of Bang
15O	Mare	2–28–23	Poll	Infected (infection of bovine origin)	— — — — —	Suspect smears

Van Volkenberg[4] studied calcification of the ligamentum nuchae of the horse. He found a small worm belonging to the Filariidae and which was similar to *Onchocerca cervicalis* in 22 ligaments out of 102 examined. Forty ligaments were cultured and *B. coli. M. pyogenes albus* and *M. pyogenes aureus* were isolated from 13. The 27 others were sterile.

Caslick[5] studied and described this parasite. He found it in 24 out of 26 ligaments. Robson[6] studied cases of filariasis of the withers in Australia. He believed that some of these infected animals develop true fistulae of the withers by "lodgement of stray pus germs on the already inflamed, worm-infested area."

Bemis,[7] in a discussion of the causes of fistulae, ascribes bruises as primarily leading to an aseptic bursitis of the supra-spinous bursa which overlies the second to the fifth thoracic spines. Bacteriological studies in these cases have shown the greater majority of cases of bursitis in this region to be sterile up to the time of perforation.

In a communication to the French Veterinary Academy, in July, 1928, Rinjard and Hilger[8] presented a report of their studies of localized pyogenic infections of equines. They studied fifteen cases of poll-evil and fistulous withers. Thirteen were observed directly and two were in the practices of their friends. It was their belief that *Bact. abortus* (Bang) of bovine origin was responsible for some of these cases of suppuration in horses. They tested the blood of supposedly normal horses for agglutinins for the Bang organism. This serum was first inactivated at 55-56°C. for forty-five minutes. One specimen out of 41 agglutinated at a titre 1-30 and one gave a partial agglutination at 1-50. As a result they believe that a serum which agglutinates at 1-50 or above should be considered as coming from a horse infected with *Bact. abortus* (Bang). Table I, translated from their article, gives some of the details of their work.

When this work came to our attention, we were very much surprised to know that the Bang organism was endemic in equines in France. The only published record of the isolation of the Bang organism from equines in this country, of which we have knowledge, is that of McNutt and Murray,[9] who have reported the isolation of the Bang organism from the aborted fetus of a mare. The first case of fistula we had an opportunity to examine was in a gelding belonging to a riding academy in Minneapolis. The attending veterinarian brought the animal to University Farm for operation. Because there was no history

TABLE II—*Results of examinations of specimens from cases of fistulae and poll-evil*

CASE	DATE (1929)	TYPE OF CASE	RESULTS		HISTORY
			BACTERIOLOGICAL EXAMINATION OF PUS	AGGLUTINATION TEST*	
1	Feb. 14	Fistula	None obtained	+ + + + + +	Recent active. Not operated
2	" 18	"	" "	+ + + — — —	Active, 18 mos. standing. Not operated
3	" 19	"	" "	+ + + + + —	5 mos. standing. Does not clear up
4	" 19	Poll-evil	" "	+ + + + + +	Active. Not operated
5	" 20	Fistula	" "	+ + + + + +	Active. Not operated
6	" 21	"	" "	+ + + + + +	Active. Not operated
7	" 23	"	" "	+ + + + + +	Active, operated and healing
8	" 23	"	" "	— — — — — —	Had fistula 4 years ago. Recovered
9	" 25	"	" "	+ + + + + +	Active
10	" 25	"	" "	+ + + + + +	"
11	" 28	"	" "	+ + + + + +	"
12	" 28	"	" "	+ + + + + +	"
13	Mar. 2	"	" "	+ + + + + +	Recovered about 4 months
14	" 2	"	" "	+ + + + + +	Recovered about 4 months
15	" 8	Poll-evil	" "	+ + + + + +	Operated about 1 month ago
16	" 14	" "	" "	+ + — — — —	Cured case
17	" 14	" "	" "	+ + + + + +	Active case
18	" 15	Fistula	" "	+ + + + — —	Active for about 2 years
19	" 20	Poll-evil	" "	+ + + + + +	Active case
20	" 21	Fistula	" "	+ + + + + +	" "
21	" 22	"	" "	+ + + + + +	" "
22	" 25	"	" "	+ + + + + P	Operated about 2 weeks ago
23	" 26	Abscess of Neck	Negative for *Bact. abortus*	No blood obtained	
24	" 27	Fistula	Negative for *Bact. abortus*	+ + + + + —	Active, small, and little exudate
25	Apr. 2	"	None obtained	— — — — — —	Active case
26	" 3	"	Negative for *Bact. abortus*	No blood obtained	Under treatment following operation 4 weeks
27	" 3	"	Negative for *Bact. abortus*	+ + + + + P	Under treatment following operation 3 weeks
28	" 4	"	None obtained	+ + + + + +	Active case
29	" 4	"	" "	+ + + + — —	Closed for 8 mos.
30	" 4	"	" "	+ + + + + +	Active case
31	" 4	"	" "	+ + + — — —	Closed for 8 mos.
32	" 4	"	" "	+ + + + + +	Active case
33	" 4	"	" "	+ + + + — —	Closed for 8 mos.
34	" 4	"	" "	+ + + + + +	Active case

TABLE II—*Continued*

CASE	DATE (1929)	TYPE OF CASE	RESULTS							HISTORY
			BACTERIOLOGICAL EXAMINATION OF PUS	AGGLUTINATION TEST						
35	" 4	"	Negative for *Bact. abortus*	+	+	+	+	+	+	Active case
36	" 9	"	Positive for *Bact. abortus*	+	+	+	+	+	+	" "
37	" 11	Abscess of Sternum	None obtained	+	+	+	+	+	+	Abscess drained
38	" 12	Fistula	None obtained	+	+	+	+	+	+	Active case
39	" 12	Poll-evil	" "	+	+	+	+	+	+	" "
40	" 12	Fistula	" "	+	+	+	+	—	—	Healed case
41	" 27	"	Negative for *Bact. abortus*	—	—	—	—	—	—	Long duration. Pus from recent pocket
42	" 29	"	None obtained	+	+	+	+	+	—	Active case
43	" 29	"	Positive for *Bact. abortus*	+	+	+	—	—	—	Recent, slight active case
44	" 29	"	Positive for *Bact. abortus*	+	+	+	—	—	—	Recent, slight active case
45	May 3	"	None obtained	—	—	—	—	—	—	Operated Nov. 26, '27. Completely recovered
46	" 9	Poll-evil	" "	+	+	+	P	—	—	Operated May, 1928. Complete recovery

*The dilutions used are 1 to 25, 1 to 50, 1 to 100, 1 to 250, 1 to 500 and 1 to 1000.
+, complete agglutination.
P, partial agglutination.
—, no agglutination.

of this animal having been in contact with cattle, we did not take a specimen of pus for bacteriological examination. We did, however, take a specimen of blood for the agglutination test, using the Bang organism for the antigen. Our surprise was great when we found that it agglutinated in a dilution in excess of 1-1,000 in 24 hours at 37° C. We were unable to secure a specimen of pus, as the animal had been taken home and the wound healed rapidly.

We immediately solicited the aid of the Minnesota veterinarians in practice in obtaining specimens of blood and pus from cases of fistula and poll-evil which they encountered. Their loyal cooperation has made this work possible.

Table II shows the results of the examinations so far conducted.

We have found positive agglutination titers in 42 cases. *Bact. abortus* (Bang) has been isolated from three specimens of pus. We have not yet determined the type of the Bang organism present.

It is evident that a large proportion of the cases of fistula and poll-evil so far studied were associated with the Bang organism. This organism evidently cannot be found very long after the fistulae are invaded by the pyogenic group of organisms. This is in keeping with its behavior in the uterus of the cow. *Bact. abortus* can and does produce suppurations in the cow. We have seen and examined subcutaneous abscesses following the injection of live culture abortion vaccine. The bacteriological examination often showed no organism except that of infectious abortion. Bacteriological studies of certain lesions of the cow's udder show them to be associated definitely with *Bact. abortus* (Bang). Also study of pyogenic lesions of the testes and seminal vesicles of the bull and boar are commonly found due to *Bact. Abortus* (Bang). It is then in keeping with the Bang organism's behavior in these animals that we should find it associated with pyogenic processes in the equine. We have no data to indicate that it is often found in the equine uterus. Further study may give us evidence of its more frequent occurrence here. Its choice of a definite location is in keeping with its definite selectivity in cattle. The gravid uterus and the udder of the female and the testes and seminal vesicles of the male are its favored spots of attack in the bovine.

Four cases so far studied do not seem to be associated with the Bang organism. This statement is based on the absence of agglutinins in the blood and the inability to isolate the germ from specimens of pus and tissue taken from the affected parts.

The presence of the germ of abortion in fistula and poll-evil explains in part the repeated observations of clinicians that these conditions were more frequently met with in country horses. These animals undoubtedly are more often in contact with cattle. The question, however, that cannot be answered at this time is through what channel does infection of the equine occur? The germ undoubtedly is taken in with the food but further experimental work is necessary to clear up this point. In some of the cases studied, it was difficult or impossible to trace direct contact with infected cows. The horse breeders of this country do not raise colts on cows' milk nor are they in the habit of feeding milk to their adult equines. It is possible that horses contract the infection in an altogether different manner than cattle.

Table III shows the agglutination titers with *Bact. abortus* antigen of blood serum from normal animals. It will be noted that four show some agglutination. These particular animals happen to be among the pure-bred horses at University Farm.

We have had an opportunity to retest their blood several times and have found the titers here reported to be constant. Physical examination fails to show any abnormalities of these animals. These findings suggest the possibility that the Bang organism may remain dormant in the body of a horse for a long time, possibly never becoming active. The large number of complete negatives, even at the 1-25 dilution, does not indicate that the blood of a horse normally agglutinates the Bang organism.

TABLE III—*Agglutination test with Bact. abortus antigen of blood serum form normal horses*

CASE	DATE (1929)	RESULTS AGGLUTINATION TEST	CASE	DATE (1929)	RESULTS AGGLUTINATION TEST
1	Apr. 13	— — — — — —	25	Mar. 8	— — — — — —
2	" 13	— — — — — —	26	" 8	— — — — — —
3	" 13	— — — — — —	27	" 8	— — — — — —
4	" 13	— — — — — —	28	" 8	— — — — — —
5	" 13	— — — — — —	29	" 8	— — — — — —
6	" 13	— — — — — —	30	" 8	— — — — — —
7	" 13	— — — — — —	31	" 8	+ + + — — —
8	" 13	— — — — — —	32	" 8	— — — — — —
9	" 13	— — — — — —	33	" 8	— — — — — —
10	" 13	— — — — — —	34	" 15	— — — — — —
11	" 13	— — — — — —	35	" 15	— — — — — —
12	" 13	— — — — — —	36	" 15	— — — — — —
13	" 13	— — — — — —	37	" 15	— — — — — —
14	Mar. 5	— — — — — —	38	" 15	— — — — — —
15	" 8	— — — — — —	39	" 15	— — — — — —
16	" 8	— — — — — —	40	" 15	— — — — — —
17	" 8	— — — — — —	41	" 15	— — — — — —
18	" 8	+ + — — — —	42	" 15	— — — — — —
19	" 8	— — — — — —	43	" 15	— — — — — —
20	" 8	— — — — — —	44	" 15	— — — — — —
21	" 8	— — — — — —	45	" 15	— — — — — —
22	" 8	— — — — — —	46	" 15	— — — — — —
23	" 8	+ + + + — —	47	" 15	— — — — — —
24	" 8	— — — — — —	48	" 15	+ + + — — —

SUMMARY

The results of this preliminary study indicate that *Bact. abortus* is associated rather closely with poll-evil and fistulae of horses. Further work is planned and under way to determine the channel of entrance of the organism and the relation of the Bang organism to other suppurative conditions (*e. g.*, so-called "cold abscesses") of equines.

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DISCUSSION

DR. N. S. MAYO: I have always wondered why they had so few cases of fistula and poll-evil in army horses. I think their record is surprisingly low, and it might be of interest to collect some data in that connection.

DR. WARD GILTNER: We have confirmed some of these results in Michigan. I would like to call the attention of this Section to paper No. 36, to be read tomorrow morning, in the Section on Poultry, covering the work of Emmel and Huddleson on the finding of this organism in poultry. It adds another species to the natural hosts of the bacillus.

CHAIRMAN BIESTER: Dr. Fitch, have you attempted to isolate the organism from the blood.

DR. FITCH: I have attempted and failed.

DR. B. T. SIMMS: I would like to call Dr. Fitch's attention to the fact that poll-evil and fistula of the withers seem to have a geographical distribution in this country not comparable with the development in cattle. In the southern states, for instance, where Bang's infection is relatively rare, fistula of the withers and poll-evil are both very common. On the other hand, in the Pacific section and the Northwest, where the infection in cattle is fairly high, poll-evil and fistula of the withers are both almost unknown. In sixteen years in that section of the country, I have seen one case of poll-evil and two cases of fistula of the withers. I think, perhaps, this phase of the distribution of the disease would give some interesting results for study.

DR. C. P. FITCH: I wish to call attention once more to those negative cases. It is not our belief for one moment that the Bang organism is the only etiological agent in fistula and poll-evil in horses.

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EXPERIMENTAL STUDIES WITH KILLED CANINE RABIES VACCINES*

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The First International Rabies Conference,[1] held in Paris, April, 1927, in its recommendations on the prophylactic vaccination of dogs, stated, among other things, that the fixed virus contained in the vaccines should be either killed or attenuated to such an extent as to be non-pathogenic for the dog when injected intramuscularly or subcutaneously. This position is well taken and the single-injection prophylactic commercial vaccines in use in this country at the present time, if prepared properly, meet this requirement in that the virus is either killed or rendered avirulent to such an extent as to be incapable of producing the disease when injected subdurally into rabbits. On the other hand, the vaccine prepared according to the method of the Japanese investigators, Umeno and Doi, does not meet this requirement,[2] as this type of vaccine has been found to be pathogenic for the dog on subcutaneous inoculation. While killed vaccines have had extensive use in this country, exact information as to their efficiency from an experimental standpoint is quite meager. Experimental work on this subject has been reported by Reichel and Schneider,[3] Schlingman[4] and the writer.[5] In the writer's reported work, evidence was obtained to indicate that in some cases phenol-killed vaccines afforded protection, in others that little protection was given by this type of vaccines against artificial infection.

In rabies experimental work difficulties are encountered in selecting a method of testing animals for immunity. Natural exposure, in which dogs are exposed to the bites of a rabid dog, at first sight may seem to furnish an ideal method of testing immunity in that, this is the method by which the disease is transmitted naturally. However, the irregularity with which normal dogs contract rabies after being bitten by rabid dogs (at least by dogs that have contracted rabies as a result of artificial exposure) makes this method unreliable. Moreover, the exposure that each

*Presented at the sixty-sixth annual meeting of the American Veterinary Medical Association, Detroit, Mich., August 13-16, 1929.

dog receives by this method cannot be regulated. One dog may receive more bites than another, the location of the wounds inflicted may be different, in some cases virus may be held back by the hair, and although wounds may be produced in these cases, little or no virus may be actually introduced into the animal. This therefore falls short of an ideal method of exposure.

The intraocular injection of street virus produces the disease in a large majority of cases, but is open to the criticism that it is too severe a test, in that infection so transmitted is much greater than would occur naturally. In the past year the intramuscular injection of street virus into the muscles of the loin has been employed and in the work about to be reported has produced the disease in a large majority of the control dogs.

J. Koch[6] has reported that injection of the virus into the muscles of the loin produces the disease with regularity. This appears to furnish a means of exposure more approximating the natural, except that the virus (in the experiments to be reported) was contained in the brain tissue, while under natural conditions it is contained in the saliva. In the following work immunity tests were made with vaccines killed or rendered avirulent by formalin, phenol and chloroform, and the methods of testing immunity were natural, intraocular and intramuscular.

Formol Vaccine (Natural Exposure)

Two dogs were vaccinated with 10 cc of a vaccine prepared as follows: Fixed rabies virus was emulsified in physiological saline solution in the proportion of 1 part of virus to 9 parts of saline. To this was added 4 per cent formalin and the mixture was allowed to stand one week. It was then centrifuged, the supernatant fluid poured off and an equal quantity of physiological saline solution was added, making the vaccine a 10 per cent suspension of the virus.

The use of a formol vaccine was first described by Plantureux[7] and it was intended to use his method of preparation. Unfortunately, through an error, 4 per cent formalin was used in the preparation of the vaccine instead of 0.4 per cent, as recommended by Plantureux. The excess of formalin, however, did not appear to cause six dogs which were inoculated any inconvenience. One of these dogs, however, developed a partial posterior paralysis eight days after injection of the vaccine. The animal showed a marked weakness and incoordination of the

posterior extremities. After several days this animal made an apparent recovery, but died on April 27, 1928.

Postmortem examination revealed an enteritis, while microscopic examination of the brain of this animal and inoculations of rabbits failed to demonstrate the presence of rabies.

The dogs were vaccinated on March 29, 1928. Two of these dogs were first exposed to the bites of a rabid dog on April 20, 1928, together with two control dogs. One vaccinated dog died of enteritis and one survived. One control dog died of rabies and one survived. The data on this experiment are contained in table I.

TABLE I—*Experiment I. Dogs vaccinated with a formalin vaccine and tested for immunity by natural exposure*

DATE (1928)	RABID DOG USED FOR EXPOSURE	DOGS EXPOSED	EXPOSURE*	RESULTS
4–20	8	1 V 2 V 3 C 4 C	Fair " " "	
4–23	6	1 V 2 V 3 C 4 C	Good " " "	Dead, April 27, 1928, not rabies Dead, May 23, 1928, rabies
5–21	4 C	2 V 3 C	Good "	
5–22	4 C	2 V 3 C	Fair Good	
5–22	12	2 V 3 C	Good Good	Alive, July 15, 1929 Alive, July 15, 1929

*Exposed to bites of a rabid dog.
V = Vaccinated.
C = Control.

Résumé: Two dogs vaccinated with a formol vaccine, together with two control dogs, were exposed to the bites of rabid dogs on different days. One vaccinated dog died of enteritis and one survived. One control dog died of rabies and one survived.

EXPERIMENT II. PHENOL-KILLED VACCINE (INTRAOCULAR EXPOSURE)

Phenol-killed vaccines were prepared from the brains of rabbits in the stage of complete paralysis as a result of subdural injection of fixed virus. One part of brain material was ground in a mortar with 4 parts of physiological salt solution containing 1 per cent phenol. This was filtered through sterile gauze and allowed to

stand at incubator temperature for 24 hours and then in the ice-box for 30 days. An equal quantity of physiological salt solution was then added, which brought the phenol content to approximately 0.5 per cent. This vaccine failed to cause the disease when injected subdurally into rabbits.

Two dogs were injected with 5 cc of vaccine on August 2, 1927, and three on March 26, 1927. On April 3, 1928, these five dogs, together with four control dogs, were given an injection of rabies street virus into the anterior chamber of the eye. Four vaccinated dogs died of rabies and one survived. All four control dogs died of rabies.

Two of the vaccinated dogs were exposed nine months after vaccination and three after about one year. The dog that survived belonged to this latter group. Table II gives the data on this experiment.

TABLE II—*Experiment II. Dogs vaccinated with phenol-killed vaccine and tested for immunity by intraocular exposure**

DOG	DATE VACCINATED (1927)	AMOUNT VACCINE (cc)	DATE OF EXPOSURE (1928)	RESULTS
5	8–2	5.0	4–3	Dead, April 24, 1928, rabies
6	8–2			Dead, April 25, 1928, rabies
7	3–26			Alive, April 1, 1929
8	3–26			Dead, April 23, 1928, rabies
9	3–26			Dead, April 26, 1928, rabies
10	Controls	——	4–3	Dead, April 24, 1928, rabies
11				Dead, April 23, 1928, rabies
12				Dead, May 28, 1928, rabies
13				Dead, April 27, 1928, rabies

*The virus consisted of dilution of 1 to 30 of rabies street virus as contained in the brain of a dog held in the ice-box 48 days. The diluent was glycerin phosphate solution and the amount used for exposure was 0.1 cc injected into the anterior chamber of the eye.

EXPERIMENT III. PHENOL- AND FORMALIN-KILLED VACCINES (INTRAOCULAR AND INTRAMUSCULAR EXPOSURE)

On October 12, 1928, three dogs were injected subcutaneously with 5 cc of a phenol-killed vaccine; three dogs with 10 cc of the same vaccine, while three dogs received 10 cc and two received 20 cc of a formalin-killed vaccine. On January 11, 1929, all eleven dogs, together with five control dogs, were exposed to rabies street virus. The exposure virus consisted of a 1-to-50 dilution* of street virus contained in the brain of a rabbit in the ice-box for 12 days.

*The diluent consisted of a glycerin phosphate solution made as follows: potassium acid phosphate, 2.5 gms.; glycerin, 500 cc; distilled water, 500 cc; adjusted after sterilization to a pH of 7.5.

One dog in each group and two control dogs were injected in the muscles of the lumbar region with 4 cc of this material. The remaining dogs were injected intraocularly with 0.1 cc of the same material.

Of the four vaccinated dogs which were exposed by intramuscular injection of street virus, three died of rabies and one survived. Those that died of rabies comprised the two phenol-vaccine animals (5-cc and 10-cc doses) and one formalin-vaccine-treated animal (10-cc dose). The dog that survived had received 20 cc of the formalin vaccine. Both control dogs in this group died of rabies. In the group exposed by intraocular injection of street virus all the animals, including the controls, died of rabies. The data on this experiment are contained in table III.

TABLE III—*Experiment III. Dogs vaccinated with various amounts of phenol- and formalin-killed vaccines and tested for immunity by intraocular and intramuscular injections of street virus**

Dog	Vaccine	Amount (cc)	Date Vaccinated (1928)	Exposure		Results
				Method	Date (1929)	
14	Phenol-killed	5.0	10–12	Intraocular	1–11	Dead, April 20, rabies
15		5.0				Dead, Feb. 11, rabies
16		10.0				Dead, Jan. 28, rabies
17		10.0				Dead, Jan. 28, rabies
18	Formalin-killed	10.0			1–11	Dead, Jan. 29, rabies
19		10.0				Dead, Feb. 6, rabies
20		20.0				Dead, Jan. 29, rabies
21	Controls	——	——		1–11	Dead, Jan. 28, rabies
22						Dead, Feb. 4, rabies
23						Dead, Jan. 26, rabies
24	Phenol-killed	5.0	10–12	Intramuscular	1–11	Dead, April 6, rabies
25		10.0				Dead, Jan. 26, rabies
26	Formalin-killed	10.0			1–11	Dead, Jan. 25, rabies
27		20.0				Alive, July 1
28	Controls	——	——		1–11	Dead, April 2, rabies
29						Dead, Jan. 28, rabies

*The virus consisted of a 1-to-50 dilution of rabies street virus contained in the brain of a rabbit held in the ice-box 12 days. The diluent was glycerin phosphate solution. 0.1 cc was injected in the anterior chamber of the eye of those dogs exposed intraocularly and 2 cc was injected in each side of the lumbar region in those dogs exposed intramuscularly.

Against intraocular exposure, the larger doses of the vaccines did not appear to afford the dogs any more protection than the smaller doses. Against intramuscular exposure the same thing held true in respect to the phenol-treated vaccine, while with the

formalin-treated vaccine, the 20-cc dose apparently protected the animal while a 10-cc dose failed. A 10-cc dose of the phenol-treated vaccine contained approximately the same amount of brain tissue as the 10-cc dose of the formalin-treated vaccine.

Experiment IV. Chloroform-Killed Vaccine (Intramuscular Exposure)

Following the successful preparation of a vaccine against rinderpest by the use of chloroform, Kelser,[8] in a preliminary report, published the results of experimental work on a rabies vaccine killed by chloroform. Rabbits, given two injections of a chloroform-killed vaccine at intervals of one week, were sufficiently immunized to withstand a subdural injection of street virus. Kelser states that the fixed virus of rabies, as contained in the vaccines, was destroyed in a few minutes by .75 per cent chloroform. This fixed virus was a strain in use in the Philippine Islands. The writer has found that the fixed virus in use in this country is more resistant to the action of chloroform. The virus in several tests was active after 11 days but not after 17 days. The method of preparation of the vaccine, which is essentially the same as that described by Kelser, except that 1 per cent chloroform is used, follows: The brain of a rabbit in the stage of complete paralysis following subdural injection of fixed rabies virus was triturated in a mortar with physiological saline in the proportion of 1 part of brain material to two parts of the saline solution.

Table IV—*Experiment IV. Dogs vaccinated with chloroform-killed vaccine and tested for immunity by intramuscular injection of street virus**

Dog	Vaccine	Amount (cc)	Date Vaccinated (1929)	Date of Exposure	Results
30	Chloroform-killed	5.0	1–22	March 22, 1929	Alive, Aug. 1
31					Dead, April 16, enteritis
32					Alive, Aug. 1
33					Alive, Aug. 1
34					Dead, April 9, enteritis
35					Dead, April 9, enteritis
36					Alive, Aug. 1
37					Alive, Aug. 1
38	Controls	——	——		Dead, April 11, rabies
39					Dead, April 5, rabies
40					Dead, April 2, rabies
41					Dead, April 9, enteritis
42					Dead, April 6, rabies
43					Killed in fight, May 15

*The virus consisted of a 1-to-50 dilution of rabies street virus contained in the brain of a rabbit which had been in the ice-box six weeks. The diluent was glycerin phosphate solution. Two cubic centimeters was injected into the muscle on each side of the lumbar region.

This was filtered through gauze and 1 per cent chloroform added. This was allowed to stand at room temperature for 24 hours and then placed in the ice-box. Subdural inoculations of rabbits showed the presence of live virus in the vaccine for 11 days. After 17 days, however, no live virus could be demonstrated. The vaccines were used about six weeks after preparation.

On January 22, 1929, eight dogs were injected subcutaneously with 5 cc of chloroform-killed vaccine. On March 22, these eight dogs, together with six control dogs, were exposed to rabies street virus by intramuscular exposure as described in previous experiments. Of the eight vaccinated dogs five survived and three died of enteritis. At this time deaths occurred among the dogs of the station due to the feeding of excessive salt meat. Microscopic examination and rabbit inoculations of the brains of the three vaccinated dogs failed to demonstrate the presence of rabies. Of the six control dogs, four died of rabies, one died of enteritis and one was killed in a fight.

Experiment V. Phenol- and Chloroform-Killed Vaccines (Intramuscular Exposure)

Table V gives the results of an experiment in which ten dogs were injected with doses of three phenol-killed vaccines ranging from 5 to 20 cc and four dogs were injected with 5 cc of a chloroform-killed vaccine. These dogs were later exposed to street virus by intramuscular injection together with nine control dogs.

Of the ten dogs treated with the phenol-killed vaccine, three died of rabies and seven survived. The three vaccinated animals that died of rabies had been treated with the same vaccine (S-3). One had received a 5-cc dose and two a 10-cc dose. While this experiment was not designed primarily to compare various lots of vaccines, it is significant to note that all animals that succumbed had received the one lot of vaccine. The four dogs treated with the chloroform-killed vaccine survived, while seven of the control dogs died of rabies and two survived.

Discussion

Of twelve dogs treated with phenol-killed vaccines and tested for immunity by intramuscular exposure, five failed to be protected. In experiment V the three animals which were treated with the vaccine S-3 were the only ones to succumb, although two dogs which had also been treated with this same vaccine survived. In experiment III, the two dogs that died had both received the same vaccine. On the other hand, of nine dogs

TABLE V—*Experiment V. Dogs vaccinated with phenol- and chloroform-killed vaccine and tested for immunity by intramuscular injection of street virus**

Dog	Vaccine	Amount (cc)	Date Vaccinated (1929)	Date of Exposure (1929)	Results
44	Phenol-killed S-1	5.0	4–1	5–14	Alive, Aug. 9†
45	Phenol-killed S-3	5.0	4–10	5–14	Alive, Aug. 9
46					Dead, June 2, rabies
47	Phenol-killed S-1	10.0	4–1	5–14	Alive, Aug. 9
48					Alive, Aug. 9
49	Phenol-killed S-3	10.0	4–10	5–14	Dead, May 28, rabies
50					Dead, June 6, rabies
51	Phenol-killed S-3	20.0	4–10	5–14	Alive, Aug. 9
52	Phenol-killed S-2	20.0	4–10	5–14	Alive, Aug. 9
53					
54	Chloroform–killed CK-1	5.0	4–10	5–14	Alive, Aug. 9
55					Alive, Aug. 9
56					Alive, Aug. 9
57					Alive, Aug. 9
58	Controls	——	——	5–14	Dead, June 24, rabies
59					Alive, Aug. 9
60					Dead, June 7, rabies
61					Dead, June 1, rabies
62					Dead, May 31, rabies
63					Dead, June 2, rabies
64					Dead, May 29, rabies
65					Alive, Aug. 9
66					Dead, June 2, rabies

*The virus consisted of a 1-to-50 dilution of street virus contained in the brain of a rabbit which had been in the ice-box two weeks. The diluent was glycerin phosphate solution. Two cubic centimeters were injected into the muscles on each side of the lumbar region.

†*Author's note:* Surviving dogs in experiments iv and v were discharged September 5 and September 17, 1929, respectfully.

treated with chloroform-killed vaccines and tested for immunity in a similar manner, none died of rabies.

The dose of a rabies vaccine is governed to a large extent by the amount of brain tissue in the vaccine, inasmuch as the virus of rabies is contained in the brain tissue. In the single-dose vaccine a relatively large amount of virus is injected at one dose, whereas in the multiple injection vaccines a number of doses of small amounts of vaccine are usually given.

The phenol-killed vaccine contains approximately 0.5 grams of fixed virus per 5-cc dose, while the chloroform-killed vaccine contains approximately 1.6 grams of fixed virus per 5-cc dose. Whether the larger amount of virus or brain tissue in the chloro-

form vaccine is responsible for the superiority of this vaccine over the phenol-killed vaccine, or whether phenol has an irregular action when used in the preparation of a killed vaccine, or both, remains to be definitely determined.

SUMMARY

The injection of rabies street virus into the muscles of the lumbar region appears to offer a satisfactory method of testing immunity against rabies in dogs.

In the limited tests made, phenol-killed vaccines, when tested for potency by artificial exposure, appeared to be variable in their action. Some lots of vaccine appeared to offer a high degree of protection, while others were lacking.

Vaccines killed with chloroform, on the other hand, rendered test animals solidly immune to artificial exposure.

The excellent results obtained with the chloroform-killed vaccines warrant a further experimental study of this method, which should be undertaken before this product is used in the field.

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Boy Scouts to Celebrate

The Boy Scouts of America will celebrate its twentieth anniversary, February 7-13, with emphasis upon its actual birthday-February 8. No better organization for boys was ever thought of than the Boy Scouts, which was started in England, in 1908, by General Lord Robert Baden-Powell, of Gilwell, the hero of Mafeking. The purpose of the movement has been character building and citizenship training for boys through activities based upon the legends of knighthood, chivalry and the lore of the plainsman and of the Indian. The imagination of boys was fired by the Baden-Powell program and three years after the movement got under way in England, the message of Scouting spread to America. Here it has grown with great rapidity and today there are nearly 825,000 boys and leaders in the United States who are actively playing the game of Scouting.

INVESTIGATIONS OF CANINE DISEASES, WITH SPECIAL REFERENCE TO RABIES

Preliminary Report*

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The increase in the number of cases of rabies in animals and the increase in the number of persons exposed during the last few years makes the question of finding a satisfactory means of controlling rabies of more serious importance to both public health officials and those whose duty it is to combat transmissible diseases of animals.

Since the Pennsylvania Bureau of Animal Industry became charged with the licensure of dogs and dog-law enforcement, the diseases of dogs have been brought more forcefully to its attention than previously, some of which are not so thoroughly understood as many diseases of other animals.

Since the "single-injection" canine rabies vaccine was first brought into use, conflicting results have been obtained by experimenters and conflicting reports have been obtained from practicing veterinarians who have used it in the field.

The Pennsylvania Bureau of Animal Industry has pursued the sane policy of not officially endorsing the use of this vaccine as a control measure, yet has not condemned it. Bureau officials have contended that we do not have enough experimental evidence to justify the Bureau in pursuing any definite policy in reference to same, and it was decided to have experiments carried out to justify the Pennsylvania Bureau in pursuing such a policy. No expense has been spared in obtaining needed buildings and equipment for the experiments which have been undertaken.

The Veterinary School of the University of Pennsylvania was invited to cooperate with the Pennsylvania Bureau of Animal Industry in carrying out these investigations on canine diseases with the special and immediate aim to test the value of com-

*Presented at the sixty-sixth annual meeting of the American Veterinary Medical Association, Detroit, Mich., August 13-16, 1929.

mercial canine rabies vaccines. Accordingly, Lentz and Barnes outlined projects which were approved and, with a few changes deemed advisable or indicated by developments, contain the outlines covering the experiments now in progress, and upon which we are rendering this preliminary report. All equipment and supplies have been furnished by the Bureau. The projects were outlined chiefly from the standpoint of experiments on canine rabies and it was a purpose to utilize the dogs in the study of other canine diseases.

The literature dealing with rabies had not been reviewed at the time these projects were outlined and has not been reviewed in detail since. We were open-minded and unbiased, and our only aim in the first of the series of experiments was to determine the merits of the single-injection canine rabies vaccines on the market and, later, to attempt to find a better product, provided that on the market was found to be lacking in immunizing properties when tested with a fair-sized dose of virus.

The projects required that an extra veterinarian be secured for carrying out the detailed examinations. Arrangements were made with Dr. A. N. Metcalfe, of the Bureau laboratory staff, to carry out these details. Metcalfe has made practically all postmortems and examinations, has made daily observations of experiment dogs, has prepared all emulsions for the inoculations, has helped make the inoculations and has carried out a number of the other details of the experiments. Lentz has cooperated in an advisory and supervisory capacity. Barnes has assumed the part of active supervision and, with the aid of Metcalfe, has prepared most of the material for this preliminary report. Practically all of the Bureau laboratory staff have cooperated in one way or another. Dr. Henry W. Turner has supervised the supply of dogs from the standpoints of feeding, care and diseases. Dr. J. G. Moon has cooperated with him in the treatments for parasites. The two Gutchall brothers have been the regular attendants and have fed and cared for the dogs in a commendable manner.

The place selected for the buildings has a southern exposure, near the foot of North Mountain, and is surrounded by enough natural shade to exclude extreme exposure to the sun. There is plenty of slope to the ground and, therefore, very good natural drainage.

The first building, which is known as the dog supply house,

was erected in 1927. It is 16 x 60 and has 6 pens, a 4-foot alley, and an outside exercise-yard at the southern end of each pen.

The kitchen, heating-plant and a small operating-room are in a building 16 x 32 and situated 12 feet west of the dog supply house.

The main canine rabies experiment building is situated 12 feet west of the operating-room and is 24 x 50. It contains 50 special woven-wire cages, the doors of which are fastened with three padlocks each. It also contains two double cages, each of which is twice the size of a single cage and has a door on each end similar to those on the front of the single cages. This double cage is known as the exposure cage and contains a trap-door which makes it possible to have two dogs in it separated or together. A rabid dog may be contained in one end while a dog is being safely placed in the other end. If the trap-door is then raised, the two dogs may occupy the same cage.

The next building, known as the observation building, is located north of the main experiment building. It is 16 x 30 and contains five 6 x 12 pens, a 4-foot alley, and an outside exercise-yard at the south end of each pen. This building is intended for keeping dogs which have passed through the early stages of experiments, under observation for prolonged periods of time and for future experimentation when required by the project or when deemed necessary, as for example, for testing the duration of immunity to rabies.

A few other buildings were necessary for hospital purposes and several isolation buildings 6 x 10 were located at places some distance from the dog supply and main experiment buildings.

The windows and doors in the main experiment building are provided with wire protection to prevent the possible escape of a dog. The spaces between buildings are protected with high wire fences and the exercise-yards of all buildings are provided with an extra wire fence some distance removed from the yard enclosures.

Two pregnant bitches were obtained from the supply house and each was placed in a separate isolation house, separated by a distance of twenty feet. They were fed and cared for by the same attendant. One of the bitches had almost recovered from a mild attack of distemper. The other was apparently healthy. The bitch showing some evidence of distemper gave birth to 6 puppies on February 7, 1929. One of these died of distemper on March 20, 1929, and later all but one died. The other bitch gave birth

to 9 puppies on March 11, 1929, all of which are alive. Some have rickets and, therefore, it cannot be said that they are healthy. Another bitch placed in the observation house gave birth to 4 puppies on April 18, 1929, and this group is still alive and healthy as far as distemper is concerned.

Distemper was quite prevalent among the various groups of dogs soon after new lots were received and has been prevalent to some extent most of the time since the first group of dogs was received yet no cases have developed in the main experiment house.

The dogs used, with few exceptions, have been ownerless dogs and, as can readily be understood, subjects suffering from various conditions and afflicted in various ways were received.

The dogs received were chiefly mongrels but dogs of all ages, types and breeds, and of both sexes were received. One can scarcely hope to collect a group of dogs of the types described without a conflagration of some sort after they are placed in a common enclosure. Men who understood and were experienced in the handling of such a collection were not available. Conflagrations which occurred in the early collections were largely prevented when groups of dogs arrived later. The two Gutchall brothers had learned how to handle groups of dogs of this type in a kindly manner. These dogs at last had found the home so much sought by every dog, whether mongrel or a representative of one of the best known breeds, and the humane act had been rendered. If any dogs received were suffering from an incurable condition or disease, the further humane act was rendered by relieving them of all suffering. In the beginning, some dogs were killed by fighters, for these were dogs which had known a life of the survival of the fittest. This was overcome in later groups by selection. Several dogs died of distemper. Mange has been the chief offender but through treatment and frequent dipping of all dogs this has been kept under control. Almost all the dogs had internal parasites which was overcome to a large degree by repeated treatments. In all, approximately 40 per cent of the first group of dogs died or were destroyed before the experiments on rabies were started. Those dogs which were left were probably as healthy a group as could be found anywhere. They were ideal for entering the projects outlined and reached their end only after they had served a useful purpose.

The first of the canine rabies experiments was not begun until all dogs remaining from the first group had been under observa-

tion for more than one year and some of them for one year and six months and this group supplied most of the dogs used in all experiments to date although a few dogs from later groups were used as controls but none were used that had not been under observation for at least six months. The dogs which died during the period of observation were autopsied and notations were made of autopsy findings. In each case an examination for rabies was made and not a single case of rabies was diagnosed among these dogs. This gave assurance that none of the dogs were exposed to rabies, at least during the observation period of more than one year, which further indicated that they were an ideal group of dogs to enter a rabies experiment.

It was necessary now to determine if a comparatively high percentage of these dogs were susceptible to rabies infection. Otherwise they would be unsatisfactory subjects for use in the experiments. The younger dogs may be the more susceptible but it was thought that the older dogs would have been afforded more opportunities to become immune, so eighteen of the older dogs were selected for the first injections of virus. It was desired through the use of these eighteen dogs, not only to estimate the susceptibility of ownerless dogs collected from various sources, but also to learn the comparative effects of different sized doses of virus and to find a convenient method of inoculation that would give uniform results in susceptible dogs.

Street virus bearing laboratory number 27993 was used for the inoculations of the eighteen dogs. This virus was obtained from the brain of a dog received for the diagnosis of rabies on November 8, 1928. It was kept in the refrigerator in 50 per cent glycerin saline until November 20, 1928, the date of the inoculations. On November 20, 25 grams of brain material was ground in a mortar and 250 cc of sterilized 50 per cent glycerin saline added to make the emulsion, after which it was filtered through four thicknesses of cheese-cloth. This would make approximately a 9 per cent emulsion, but if allowance is made for glycerin which adhered to bits of brain material used and that removed by filtering, the final emulsion would probably not be more than 7 to 8 per cent brain material, and just how much virus it contained would have been more difficult to determine. On the basis of 8 per cent, the smallest dose of the emulsion used would contain less than one-third of a minim of brain material. The method of preparation of the emulsion of brain material is described somewhat in detail here, because the same method has been adhered to throughout,

with the exception that in the preparation of the emulsions used later, 2 grams of brain material was emulsified in 20 cc of the 50 per cent glycerin saline, which is in the same proportion but in smaller amounts. The emulsions used, however, are referred to throughout this report as 10 per cent emulsions.

The 18 dogs were divided into six groups, consisting of three dogs each. One dog from each group was inoculated on November 20, 1928, with 0.25 cc of the emulsion, one with 0.5 cc and one with 1.0 cc.

The first group of three dogs was inoculated subcutaneously medial to the radio-ulnar articulation and an attempt was made to injure the median nerve with the needle point: the second group was inoculated intramuscularly, in the pectoral region; the third intraperitoneally; the fourth intraocularly; the fifth intravenously over the medial carpal region; and the sixth was given a

TABLE I—*Group 1. Virus 27993 injected subcutaneously, November 20, 1928*

Dog	Virus	Exposure		Dose (cc)	Died	Remarks
		Date	Method			
41	27993	11-20-28	Subcutaneous	0.25		
	27995-1	4-5-29	Subarachnoid	0.25	12 days	Rabies
52	27993	11-20-28	Subcutaneous	0.5		
	29995-13	5-14-29	Subarachnoid	0.25		
	29995-81	7-12-29	Subarachnoid	0.25		Alive, 8-10-29
55	27993	11-20-28	Subcutaneous	1.0		
	29995-1	4-5-29	Subarachnoid	0.25	13 days	Rabies

TABLE II—*Group 2. Virus 27993 injected intramuscularly, November 20, 1928*

Dog	Dose (cc)	Died	Remarks
108	0.25	40 days	Rabies
101	0.5	29 days	Rabies
18	1.0	31 days	Rabies
Average		33½ days	100%

TABLE III—*Group 3. Virus 27993 injected intraperitoneally, November 20, 1928*

Dog	Dose (cc)	Died	Remarks
33	0.25	47 days	Rabies
27	0.5	25 days	Negative
34	1.0	21 days	Rabies
Averages	3 dogs	31 days	100%
	2 dogs (rabies)	34 days	67%

subarachnoid inoculation in the atlanto-axial region. The results of the inoculations of the six groups are shown in the accompanying tables.

TABLE IV—*Group 4. Virus 27993 injected intraocularly, November 20, 1928*

DOG	DOSE (CC)	DIED	REMARKS
96	0.25	26 days	Rabies
75	0.5	17 days	Rabies
65	1.0	Killed 80 days	Vicious dog
Average		21½ days	67%

TABLE V—*Group 5. Virus 27993 injected intravenously, November 20, 1928*

DOG	DOSE (CC)	DIED	REMARKS
25	0.25	30 days	Rabies
95	0.5	39 days	Rabies
21	1.0	40 days	Rabies
Average		36½ days	100%

TABLE VI—*Group 6. Virus 27993 injected subarachnoidly, November 20, 1928*

DOG	DOSE (CC)	DIED	REMARKS
17	0.25	20 days	Rabies
29	0.5	25 days	Rabies
100	1.0	17 days	Rabies
Average		20½ days	100%

Summary of dogs inoculated 11-20-28 with street virus 27993

3 dogs inoculated subcutaneously did not die until after a second exposure to another method. One was still alive, August 10, 1929.
3 dogs inoculated intravenously died in an average of 36½ days.
3 dogs inoculated intramuscularly died in an average of 33½ days.
3 dogs inoculated intraperitoneally died in average of 31 days.
3 dogs inoculated intraocularly, 2 died in an average of 21½ days.
3 dogs inoculated subarachnoidally died in an average of 20½ days.
A total of 14 dogs died in an average of 29 days.

(Rabbits were inoculated as controls in the use of virus 27993 and for diagnostic purposes when there was any question as to the diagnosis. These are shown in table VII.)

Our results seem to indicate that peripheral nerve trunks are not suitable channels for the inoculation of rabies virus and that intravenously is a very good method but that the period of incubation following intravenous inoculation is longer than that following subarachnoid inoculation. Different areas for inoculation in nerve trunks and different nerves should be tried.

TABLE VII—*Rabbit inoculations with virus 27993**

GROUP	RABBIT	EXPOSURE		DIED	REMARKS
		DATE	METHOD		
C†	7189	11–20–28	Intraocular	21 days	Rabies
	7190			86 days	Rabies
C	7197	11–22–28		22 days	Rabies
	7198			37 days	Rabies
D-34	7256	12–21–28		16 days	Rabies
	7257			16 days	Rabies
D-27	7258	12–21–28			Killed
	7259				Alive§
D-96	7260	12–21–28	Intracranial		Killed
	7261				Alive§
D-25	7288	12–28–28		206 days	Rabies
	7289			17 days	Rabies
D-101	7290	12–28–28		37 days	Rabies
	7291			20 days	Rabies
C	7500‡	2–21–29			Alive§
	7501				Alive§

*0.25 cc in all cases.
†C = virus control, D = diagnostic; the numbers following D indicate the dogs from which the virus was secured.
‡Rabbits 7500 and 7501 were inoculated with an emulsion prepared on November 20, 1928, which was the same emulsion as that used for rabbits 7189, 7190, 7197 and 7198.
§August 10, 1929.

None of the three dogs inoculated subcutaneously died of rabies for a period of 150 days following the subcutaneous inoculations. Either we were unsuccessful in injuring the median nerve or the large nerve trunks are not suitable places for infecting animals with the virus of rabies. Nerve trunks are commonly referred to as being very receptive channels. The deaths of two dogs after the 150th day were undoubtedly due to the second exposures through another channel on April 5, 1929.

On the contrary, all dogs inoculated intravenously died of rabies. In our intravenous inoculations a blood-vessel running very close to the skin was selected. The needle reached the interior of the vessel usually with the first thrust. No virus was introduced until blood was running freely from the needle. Probably most of the virus injected had been carried away by the blood before the needle was withdrawn, and there would be very little chance for sufficient virus to be deposited in the region of nerves from what would leak from the vessel after the needle was withdrawn. A twenty-gauge needle was used and very little

bleeding through the skin occurred following inoculations, and no noticeable swellings occurred under the skin.

Virus 27993 was somewhat slower in producing its effects than some viruses used later.

The subarachnoid method of inoculation was decided upon for most later exposures, because one is more sure that the inoculation is properly done, and it is also a convenient method for obtaining uniform results and aside from causing rabies apparently causes no other harmful effects in animals inoculated, except when badly contaminated viruses are used. The 0.25 cc of the emulsion used seemed to contain sufficient virus to produce the disease in susceptible animals and, therefore, was the regulated dosage decided upon for the exposures in the first of the series of vaccination experiments, although other strains of virus were used for these exposures. We believe it possible for dogs to receive natural exposures equal to that represented by this dose.

The single-injection-method canine rabies vaccines were obtained from four different commercial laboratories, which we consider among the best in the country, and were used as nearly as possible in accordance with directions contained on or accompanying the packages. The prescribed dose of vaccine was injected subcutaneously in the lateral neck region anterior to the shoulder.

The four vaccines for convenience and to avoid showing the identity of the laboratories from which obtained were classified as A, B, C and D.

Ten dogs were inoculated with each of the four vaccines. We had previously gained the impression that immunity was supposed to be fully established in 21 days following vaccination. No dogs were exposed until 36 days subsequent to vaccination. This happened to only 2 of the dogs vaccinated with vaccine A. Four dogs in group A were first exposed at 39 days, 1 at 57 days, 2 at 70 days and 1 at 155 days. Dogs in groups B, C and D were first exposed: 2 at 42 days, 4 at 45 days, 1 at 63 days, 2 at 76 days and 1 at 161 days. The reason for the difference in A and the other groups is that the group of dogs receiving vaccine A were inoculated 6 days later than the other groups. Dogs which lived after the first exposure were exposed again in around 60 days and some were exposed even the third time at 60 days subsequent to the second exposure.

We tried to make things as nearly equal as possible in all four groups, both in the selection of dogs and viruses used. We over-

TABLE VIII—*Tests of vaccine A—10 dogs inoculated, February 7, 1929*

Dog	Approximate Age	Sex	Exposures After Vaccination				Died	Remarks
			Days	Virus	Dose (cc)	Method		
7	2 years	F	36	29870	0.25			
			96	29995–13	0.25	Subarachnoid		
			155	29995–81	0.25		16 days	Rabies
15	2 years	F	39	29952	0.25	Intraocular		
			96	29952–171	0.25	Subarachnoid	15 days	Rabies
23	7 years	M	70	29995–64	?	Dog 64		Alive*
37	3 years	F	155	27037–7945R	0.25	Intravenous		Alive*
76	2 years	M	70	29995–139	?	Dog 139		Alive*
77	2 years	M	39	29995	0.25			
			96	29995–13	0.25		17 days	Negative?
87	4 years	M	39	29995	0.25	Subarachnoid	17 days	Rabies
88	4 years	M	39	29952	0.25		4 days	Septicemia
92	3 years	M	57	29995–1	0.25		15 days	Rabies
104	Aged	F	36	29870	0.25		18 days	Rabies

*August 10, 1929.

TABLE IX—*Tests of vaccine B—10 dogs inoculated, February 1, 1929*

Dog	Approximate Age	Sex	Exposure After Vaccination				Died	Remarks
			Days	Virus	Dose (cc)	Method		
36	6 years	F	45	29952	0.25	Subarachnoid	21 days	Rabies
39	3 years	F	76	29995–139	?	Dog 139		Alive*
40	5 years	F	42	29870	0.25			
			102	29995–13	0.25	Subarachnoid		
			161	29995–81	0.25		16 days	Rabies
43	2 years	F	76	29995–64	?	Dog 64		Alive*
44	3 years	F	42	29870	0.25			
			102	29995–13	0.25			
			161	29995–81	0.25			Alive*
						Subarachnoid		
50	5 years	F	45	29995	0.25		17 days	Rabies
53	3 years	F	45	29995	0.25		20 days	Rabies
54	3 years	F	45	29952	0.25	Intraocular		
			102	29952–171	0.25	Subarachnoid	15 days	Rabies
56	5 years	F	161	27039–R7945	0.25	Intravenous		Alive*
59	6 years	F	63	29995–1	0.25	Subarachnoid	20 days	Rabies

*August 10, 1929.

TABLE X—*Tests of vaccine C—10 dogs inoculated, February 1, 1929*

Dog	Approximate Age	Sex	Exposure After Vaccination				Died	Remarks
			Days	Virus	Dose (cc)	Method		
11	4 years	F	45	29952	0.25	Intraocular		
			102	29952–171	0.25	Subarachnoid	17 days	Rabies
26	Aged	M	63	29995–1	0.25	Subarachnoid	12 days	Rabies
28	5 years	M	76	29995–64	?	Dog 64		Alive*
31	6 years	M	76	29995–139	?	Dog 139		Alive*
51	3 years	F	45	29952	0.25			
			102	29952–171	0.25	Subarachnoid		
			161	29995–81	0.25			Alive*
58	5 years	F	161	27039–R7945	0.25	Intravenous		Alive*
66	3 years	M	42	29870	0.25			
			102	29995–13	0.25		22 days	Rabies
72	3 years	M	45	29995	0.25	Subarachnoid	19 days	Rabies
86	3 years	M	42	29870	0.25		16 days	Rabies
107	4 years	F	45	29995	0.25		19 days	Rabies

*August 10, 1929.

TABLE XI—*Tests of vaccine D—10 dogs inoculated, February 1, 1929*

Dog	Approximate Age	Sex	Exposure After Vaccination				Died	Remarks
			Days	Virus	Dose (cc)	Method		
8	3 years	F	45	29952	0.25			
			102	29952–171	0.25		14 days	Rabies
13	5 years	F	45	29995	0.25	Subarachnoid	14 days	Rabies
63	4 years	M	161	27039–R7945	0.25	Intravenous		Alive*
64	7 years	M	63	29995–1	0.25		17 days	Rabies
78	5 years	M	45	29995	0.25	Subarachnoid	15 days	Rabies
80	Aged	M	45	29952	0.25	Intraocular		
			102	29952–171	0.25	Subarachnoid	14 days	Rabies
81	3 years	M	76	29995–64	?	Dog 64	73 days	Rabies
84	3 years	M	42	29870	0.25		25 days	Rabies
90	5 years	M	42	29870	0.25	Subarachnoid	21 days	Rabies
91	3 years	M	76	29995–139	?	Dog 139		Alive*

*August 10, 1929.

looked the sexes and did not get an equal number of the two sexes in each group but we believe this made no difference in the results obtained. The results obtained, as far as exposures have been been made, are shown in the accompanying tables.

TABLE XII—*Results of inoculation of 16 control dogs*

Dog	Approximate Age	Sex	Exposure				Died	Remarks
			Virus	Date (1929)	Dose (cc)	Method		
1	2 years	F	29995	3–18	0.25		15 days	Rabies
2	2 years	F	29870	3–15	0.25		19 days	Rabies
9	2 years	F	29952	3–18	0.25	Subarachnoid		
			29952–171	5–14	0.25		13 days	Rabies
10	3 years	F	29952	3–18	0.25	Intraocular		
			29952–171	5–14	0.25	Subarachnoid	15 days	Rabies
14	3 years	F	29995–1	4–5	0.25		13 days	Rabies
16	2 years	F	29995–1	4–5	0.25		18 days	Rabies
105	2 years	F	29995	3–18	0.25		18 days	Rabies
106	2 years	F	29995–1	4–5	0.25	Subarachnoid		
			29995–13	5–14	0.25			
			29995–81	7–12	0.25			Alive†
109	2 years	F	29870	3–15	0.25		17 days	Rabies
141	3 years	M	29995–139	4–18	?	Dog 139		Alive†
171*	5 years	F	29952–36	4–19	0.25	Subarachnoid	19 days	Rabies
198		M	29995–64	4–18	?	Dog 64		Alive†
211		M	29995–139	4–18	?	Dog 139		Alive†
239*	3 years	M	29952–36	4–19	0.25	Subarachnoid	55 days	Rabies
224	3 years	M	27039–R7945	7–12	0.25		24 days	Rabies
231	Aged	M	27039–R7945	7–12	0.25	Intravenous		Alive†

*Dogs 171 and 239 received virus from dog 36 and possibly should not be included in the control group because none of the vaccinated dogs received this virus from dog 36, although they did receive the same strain at the same time that dog 36 received it and again after passage from dog 36 through dog 171.

†August 10, 1929.

Rabbits were also used as controls. The results of the rabbit inoculations are shown in table XIII.

In the case of each dog of the vaccinated or control groups which died following exposure, if there were any question as to the diagnosis, rabbits were inoculated. Table XIV shows the

number of rabbits used for this purpose and the results of the inoculations.

Table XV, showing in condensed form the results after exposure of vaccinated dogs, control dogs and control rabbits, does not contain those exposed to rabid dogs and does not contain the dogs exposed intravenously on July 12, 1929.

TABLE XIII—*Control rabbits inoculated with viruses used for exposing vaccinated and control dogs*

RABBIT	VIRUS	DATE (1929)	DIED	REMARKS
7594	29870	3–15	77 days	Rabies
7595	29870	3–15	27 days	Rabies
7605	29952	3–19	——	Alive, 8–10–29
7606	29952	3–19	1 day	Decomposed virus
7607	29995	3–19	1 day	Decomposed virus
7608	29995	3–19	15 days	Rabies
7670	29995	4–8	22 days	Rabies
7671	29995	4–8	17 days	Rabies
7727	29952–36	4–20	25 days	Rabies
7728	29952–36	4–20	——	Alive, 8–10–29
7992	29870–2	7–19		Alive, 8–10–29
7993	29870–2	7–19		Alive, 8–10–29
7994	29870–90	7–19		Alive, 8–10–29
7995	29870–90	7–19		Alive, 8–10–29

TABLE XIV—*Diagnostic rabbits inoculated intracranially with viruses used for exposing vaccinated and control dogs*

DOG	RABBIT	VIRUS*	DIED	REMARKS
50	7681	29995	25 days	Rabies
50	7682	29995	25 days	Rabies
55	7739	29995–1	31 days	Rabies
55	7740	29995–1	53 days	Rabies
77†	7872‡	29995–13	——	Alive§
77	7873‡	29995–13	——	Alive§
11	7874	29952–171	14 days	Rabies
11	7875	29952–171	21 days	Rabies
R7875	7935	29952–171	13 days	Rabies
R7875	7936	29952–171	22 days	Rabies

*0.25 cc in all cases.
†Dog 77 was classified in the vaccine A chart as negative(?) to rabies.
‡Inoculated June 6, 1929.
§August 10, 1929.

DISCUSSION

The totals would seem to indicate that, based on our tests, the vaccine of any one of the laboratories is not any better and not any worse than that of any of the other three and, that the vaccinated dogs were not any more immune to rabies than the controls. The one dog in group A died of septicemia, which leaves only six, and one of these which died was not definitely proved to

TABLE XV—*Summary of data on vaccinated and control dogs*

GROUP	EXPOSURES						TOTALS				RABIES		
	FIRST		SECOND		THIRD			DIED					
	No.	DIED	No.	DIED	No.	DIED	EXPOSURES	No.	%	ALIVE	POS.	?	NEG.
A	7	4	3	2	1	1	7	7	100	0	5	1	1
B	7	4	3	1	2	1	7	6	85	1	6	0	0
C	7	4	3	2	1	0	7	6	85	1	6	0	0
D	7	5	2	2	0	0	7	7	100	0	7	0	0
Total	28	17	11	7	4	2	28	26	92	2	24	1	1
Controls	11	8	3	2	1	0	11	10	91	1	10	0	0
Total	39	25	14	9	5	2	39	36	91	3	34	1	1
Rabbit Controls	10*	8					10	8	80	2	6	0	2†

*Exposed only once.
†Died the day following the inoculation of virus.

have rabies. Considering only the definite cases of rabies, the septicemia dog excluded, we have the following summary:

Of 27 dogs vaccinated and later exposed, 24 (89 per cent) died of rabies.

Of 11 unvaccinated controls exposed, 10 (91 per cent) died of rabies.

During all the time since these experiments were begun, susceptible dogs have been kept in the experiment house in close contact with rabid dogs as cases developed or in the same cages previously occupied by rabid dogs and no cases of rabies have developed in them.

All vaccinated dogs remained healthy until after exposure to virus. Apparently the use of vaccine causes no harmful effects.

Dogs placed in the individual cages kept in noticeably better physical condition than those confined in the common enclosures of the dog supply house.

All of the results reported here are based on things which actually happened; and, therefore, are based on facts. These results probably differ to a considerable degree from those reported by others.

There seemed to be no indication that the vaccines used had the power of immunizing dogs against any of the viruses used. Neither was there any indication that one injection of virus immunized against a subsequent injection, sixty or more days later, of the same strain or a different strain of virus. If strains of virus are in existence that, by a single-injection method in the

dosage now recommended, will immunize dogs against subsequent exposures to fresh street virus, our results would seem to indicate that none of the four laboratories is now using such a strain.

The chief method used by us in exposing these dogs did not exactly represent natural exposure although it did, we believe, represent what may occasionally take place from natural exposures, and, from the standpoint of getting the virus inoculated into a receptive channel, we believe this method represents what must take place from natural exposure if a high percentage of cases of rabies is to follow the exposure. It is possible to get equal exposure with few exceptions by the subarachnoid method. In a few cases it is not possible to know that the injections are good. On the other hand, it is impossible to get equal exposures through exposing to a rabid dog. Possibly not over 20 to 30 per cent of dogs bitten by rabid dogs develop rabies. To depend on such a method would seem a great sacrifice of dogs which could be used to better advantage and, then, it would be necessary to draw uncertain conclusions. It is not possible to expose and get equal biting even if a group of several dogs are placed in a common enclosure with a rabid dog. The biting period of a rabid dog is limited. More humane measures can be practiced by exposing in other ways. Of the eleven dogs shown in the tables, which were exposed to a rabid dog, only one died of rabies and this was a vaccinated dog.

Source of Viruses

The original virus of each virus used in these experiments was obtained from brain material sent to the laboratory for the diagnosis of rabies. The only requirement was that Negri bodies be found in the material received for diagnosis. Viruses 26959 and 27039 are being successively passed through rabbits for the purpose of securing one or two fixed viruses. They have now passed through 18 rabbits. The history of all viruses used is briefly shown below.

No. 26959. Ox brain. Received August 27, 1928, from Pittsburgh, Penna. No information.

No. 27039. Dog brain. Received September 4, 1928, from Wayne, Penna.

No. 27993. Dog brain. Received November 8, 1928, from Lansdowne, Penna.

No. 29870. Dog brain. Received March 8, 1929, from Collegeville, Penna. Bitten by stray dog three weeks prior to

death. Stray dog had also been diagnosed positive to rabies by Pa. B. A. I. laboratory.

No. 29952. Dog brain. Received March 14, 1929, from Royersford, Penna. Believed to have been bitten by a rabid dog 17 days before death.

No. 29995. Dog brain. Received March 16, 1929, from Oxford, Penna. Man bitten March 14, 1929. Dog found dead, March 16, 1929.

The results obtained with some viruses were of considerable interest. Table XVI illustrates what may happen following the use of a decomposed virus (29952).

TABLE XVI—*Virus 29952 (decomposed)—10% emulsion (0.25 cc in all cases)*

Dog	Group	Virus	Exposure		Died	Remarks
			Date (1929)	Method		
8	D	29952	3–18			
		29952–171*	5–14	Subarachnoid	14 days	Rabies
9	Control	29952	3–18			
		29952–171	5–14		13 days	Rabies
10	Control	29952	3–18	Intraocular		
		29952–171	5–14	Subarachnoid	15 days	Rabies
11	C	29952	3–18	Intraocular		
		29952–171	5–14	Subarachnoid	17 days	Rabies
15	A	29952	3–18	Intraocular		
		29952–171	5–14	Subarachnoid	15 days	Rabies
36	B	29952	3–18		21 days	Rabies
51	C	29952	3–18	Subarachnoid		
		29952–171	5–14			
		29995–81†	7–12			Alive, 8-10-29
54	B	29952	3–18	Intraocular		
		29952–171	5–14	Subarachnoid	15 days	Rabies
80	D	29952	3–18	Intraocular		
		29952–171	5–14	Subarachnoid	14 days	Rabies
88	A	29952	3–18		4 days	Septicemia
171	Control	29952–36‡	4–19	Subarachnoid	19 days	Rabies
239	Control	29952–36	4–19		55 days	Rabies

*Obtained from dog 171.
†Obtained from dog 81.
‡Obtained from dog 36.

Note: Only one of the 10 dogs injected with street virus 29952, on March 18, 1929, died of rabies, and this was a vaccinated dog.

Three of the dogs became very sick within a day following the inoculation and dog 88 died on the fourth day, of septicemia. The others made a complete recovery from this temporary illness. This virus 29952 was rather badly decomposed, which we believe was responsible for its killing only one dog (36) of rabies. Virus from dog 36 was inoculated into dogs 171 and 239, with the result that both died of rabies. The eight other dogs were in turn inoculated with virus from 171, with the result that seven of the eight died of rabies in an average of slightly less than 15 days. Had no decomposed viruses been used for exposing vaccinated and control dogs, the condensed table of results would undoubtedly show fewer second and third exposures.

TABLE XVII—*Virus 29870 (decomposed)—10% emulsion (0.25 cc in all cases, subarachnoid inoculation)*

DOG	GROUP	VIRUS	EXPOSED (1929)	DIED	REMARKS
2	Control	29870	3–15	19 days	Rabies
7	A	29870	3–15		
		29995–13*	5–14		
		29995–81†	7–12	16 days	Rabies
40	B	29870	3–15		
		29995–13	5–14		
		29995–81	7–12	16 days	Rabies
44	B	29870	3–15		
		29995–13	5–14		
		29995–81	7–12		Alive, 8-10-29
66	C	29870	3–15		
		29995–13	5–14	22 days	Rabies
84	D	29870	3–15	25 days	Rabies
86	C	29870	3–15	16 days	Rabies
90	D	29870	3–15	21 days	Rabies
104	A	29870	3–15	18 days	Rabies
109	Control	29870	3–15	17 days	Rabies

*Obtained from dog 13.
†Obtained from dog 81.

Virus 29870 was also somewhat decomposed which may account for its causing rabies in only 6 of the 10 dogs injected on March 15. These 6 dogs died in an average of 19 days.

VIRUS 29995

Twenty-three dogs exposed to a subarachnoid injection of

virus 29995 died in an average of 16 plus days, one of which required the second exposure, and in two the diagnosis of rabies was not confirmed by the finding of Negri bodies. One dog which was a control did not succumb after the third exposure. Of 11 dogs exposed to rabid dogs, one died of rabies in 73 days. This was a vaccinated dog. These rabid dogs were probably too far advanced and were not good biters. They tried to bite but apparently had lost partial control of the lower jaw.

Fresh Viruses

Fresh viruses which have not undergone decomposition have apparently produced the most uniform results. Four unvaccinated dogs inoculated (subarachnoid) on February 14, 1929, with emulsions of viruses 26959 and 27993 prepared November 20, 1928, and kept in the refrigerator, did not succumb to rabies. These four dogs were exposed to a subarachnoid injection of virus 29995 on May 14, 1929, and the four died of rabies following these exposures, one in 19 days, two in 18 days, and the other one in five days. The dog that died in five days showed typical symptoms the second day after this (May 14) exposure and there is the possibility that the virus used on February 14 had been sufficiently attenuated to cause a longer incubation period. Some other dogs, exposed to virus 29995, however, showed symptoms as early as the ninth day. We believe a larger dose of viruses kept over different periods of time is required than when fresh virus is used. There is some evidence to indicate that with equal dosage the period of incubation is longer when viruses are kept in the refrigerator for some time than when fresh viruses are used.

Concluding Statements

There has possibly been a greater increase in rabies throughout the country, both in animals and humans, since the single-dose rabies vaccine came into use than for any other period known. Yet the states are better equipped with transmissible animal disease control organizations. This increase may be due to a feeling of false security built up through the use of the single-injection vaccine.

In the preparation of this preliminary report we have purposely avoided commenting on, or referring to, the work done by others. We hope to give a more detailed report of further developments later.

We have aimed in this report to give the reader a fair picture of the equipment and the manner in which the experiments have

been conducted to date but probably some necessary details have been left out.

In future reports it will not be necessary to describe the grounds, buildings and equipment.

Conclusions will not be drawn until a later report.

We are open to suggestions for the benefit of the future of this work. We aim to determine if a suitable immunizing agent can be developed.

ACKNOWLEDGMENTS

The writers wish to express their appreciation to all those who cooperated in making this project possible as well as to those who have actively cooperated during the progress of the experiments. We especially express our appreciation and extend our thanks to the laboratories which furnished the vaccines for the first of the series of experiments.

Have you sent in your subscription to the International Veterinary Congress? See page 542, the JOURNAL, November, 1929.

Veterinary School, Hanover Germany.

THE CONTROL OF RABIES IN PENNSYLVANIA*

By T. E. MUNCE, *Harrisburg, Pa.*

Director, Pennsylvania Bureau of Animal Industry

I hesitate to discuss the subject assigned me, "The Control of Rabies in Pennsylvania," for the reason that I do not feel that I can offer anything new or of special interest in the control of rabies. However, your Section Secretary, Dr. Brown, rather insisted upon my telling concerning the method by which we control rabies in Pennsylvania and I felt that it was my duty to do so.

Pennsylvania has an area of 45,215 square miles and a population of something over 10,000,000 people, or 225 persons to each square mile. We have approximately 700,000 dogs in Pennsylvania, or 16 dogs per square mile.

Our purpose in controlling rabies is to protect our people and their property against damage by rabid animals. Cases of rabies are usually reported by the private veterinarians, although local Health Board officials and others frequently report cases to us. Usually the brain of the suspected dog is submitted to the Laboratory of the Bureau of Animal Industry. If the laboratory report is positive or if the case is diagnosed in the field by the private practitioner as positive, the Bureau of Animal Industry establishes a special quarantine against all animals known or suspected to have been bitten by the rabid animal. The quarantined animal must be penned up or kept tied for a period of 100 days, or until such time as the order is revoked. In most instances, a special quarantine is all that is required to control an outbreak of rabies.

Occasionally we experience a widespread outbreak, with a large number of dogs involved, and wherein the special order of quarantine is not sufficient to control the situation. In such cases, we resort to a general rabies quarantine.

A special quarantine may be established and enforced by an agent of the Bureau of Animal Industry, whereas a general quarantine must be established by the Department of Agriculture, and is enforced by the agents of the Bureau of Animal Industry.

In 1927, we had three general quarantines against rabies: one in Pittsburgh, and which involved the thickly populated section

*Presented at the sixty-sixth annual meeting of the American Veterinary Medical Association, Detroit, Mich., August 13-16, 1929.

of that city of close to 800,000 people; another in Wilkes-Barre, which is a city of approximately 100,000 people; and another, covering all of the county of Delaware, which county is adjacent to Philadelphia, and is thickly populated. In the year 1928, we had but one general quarantine, in the city of Pottsville. We have not had an occasion to establish a general rabies quarantine so far this year.

The law provides that when a general rabies quarantine is established, notice must be posted. We usually post a large number of notices in order to give the public notice that a quarantine exists and concerning its requirements. In addition to posting notices, the law provides that the order of quarantine shall be advertised in one newspaper. We usually advertise in several newspapers circulating generally within the quarantined territory. After a general quarantine is established, it is unlawful for any dog to be at large unless it is effectually muzzled to prevent biting or unless it is on leash. All other dogs, whether licensed or not, may be shot.

We provide a small Ford truck, equipped with a cage which is operated by two men who are carefully selected for the work. In open territory, the dogs are shot, the bodies placed in the truck and disposed of locally. In thickly populated sections, where the discharge of a gun is not deemed expedient, the outlaw dogs are taken up and disposed of with the cooperation of the local humane society.

In enforcing general quarantines, we cooperate with the local health authorities. Needless to say, we always work hand-in-hand with the local practicing veterinarians, so that when we establish a quarantine and undertake to enforce it, we have the united support of all of the local agencies, including the police department. Now, if the quarantined area is a large one, we increase our enforcement machinery in proportion to the amount of territory to be covered and the work to be done.

We have been handling rabies under this method since the State Live Stock Sanitary Board, which is now the Bureau of Animal Industry, was established in 1895, a period of about thirty-four years. Our method has proved successful. In fact in but one case were we obliged to extend a period of general quarantine from the length of time originally established—100 days.

In addition to enforcing the rabies quarantine, the enforcement of the Pennsylvania Dog Law has been assigned to the Pennsyl-

vania Bureau of Animal Industry and we have a Dog Law Enforcement Division within our Bureau. The State is divided into seventeen districts at present, with a suitable man in charge of the dog-law-enforcement work in each district. These dog-law-enforcement agents assist in the enforcement of rabies quarantines, at least to the extent of seeing as best they can that all dogs are licensed and under proper control.

You may be interested to know that, during the year 1927, we had 210 cases of rabies reported; 512 animals and 173 persons were bitten: 632 individual quarantines were established over the bitten and exposed animals; and 348 dogs were killed. In the year 1928, we had 214 cases, 2,281 animals and 172 persons were bitten; 2,601 animals on 546 premises were quarantined; and 398 animals were killed. Up to July of this year, a period of six months, 24 cases were reported, 60 animals and 13 persons were bitten; 63 animals on 34 premises were quarantined; and 42 dogs were killed.

From January 1, 1927, to August 1 of this year, a period of two years and seven months, 1,167 brains were examined for rabies at the Laboratory of the Bureau of Animal Industry. Of this number, 640 were positive.

I have endeavored to give you, as briefly as possible, the method which we follow in Pennsylvania in our efforts to control rabies.

Six hundred forty positive cases out of approximately 700,000 dogs, which is about nine ten-thousandths (.0009) of one per cent, over a period of two years and seven months, is not a bad record in controlling rabies in Pennsylvania. This, plus the fact that over a period of thirty-four years on but one occasion have we been obliged to extend a quarantine, justifies the statement that our quarantine method for controlling rabies in Pennsylvania has been successful. In this work, the Bureau has been supported by the veterinary profession throughout the State, by the State Department of Health, local health boards and, in fact, all cooperating agencies have supported our efforts splendidly. We feel that we have been getting along so satisfactorily, that until a better method is put forth, we should continue the quarantine method which we are using at the present time. However, if a better method is advanced, we will be very willing to consider it.

We shall gladly furnish copies of our laws, quarantines and other information covering the control of rabies to those interested.

DISCUSSION

DR. C. P. FITCH: I would like to ask Dr. Munce one or two questions about his paper. First, I would like to know what constitutes a rabies quarantine in these general quarantined districts, like the city of Pittsburgh. He said nothing about muzzling the dogs, he said nothing about confining the dogs, he said nothing as regards what constitutes the quarantine.

The other question is making the diagnosis of rabies, upon what does he base the diagnosis? In other words, finding Negri bodies, or inoculation of animals, or both?

DR. MUNCE: I am going to answer Dr. Fitch's question by reading that part of the Pennsylvania Quarantine Law which defines the manner in which dogs may be handled under a general rabies quarantine. I quote:

"It shall be unlawful, after notice as aforesaid, for the owner of any dog to permit such dog to run at large in any such quarantined locality, unless such dog shall be muzzled so as to effectually prevent biting; or for any person to remove, or permit to be removed, any dog from such quarantined area. Any dog found running at large in such quarantined area, or known to have been removed from such area, not being muzzled as aforesaid, may be secured and confined, or may be shot or otherwise destroyed, by any person, without liability therefor."

We do not in all cases shoot unmuzzled dogs. For illustration, in the city of Pittsburgh, arrangements were made with the Animal Rescue League to take up the unmuzzled dogs which were collected by our quarantine enforcement men and we turned over to the League, a rather large number of dogs, many of which were licensed, placed in good homes and well cared for. The balance were humanely killed by the League. In localities where some such similar arrangements cannot be made, the unmuzzled dogs are shot by the State Quarantine Enforcement officers or are caught alive and placed in a steel tank which we had especially made for the purpose. This tank was placed on the rear of a Ford truck and the exhaust of the motor piped into the tank with the result that the dogs were asphyxiated. This arrangement had the approval of the Humane authorities.

It is rather difficult for me to attempt to tell you in detail the manner in which all cases are handled. The big point in eradicating an outbreak of rabies is to control the dogs. With the dogs under control, we can eradicate rabies.

I am going to ask Dr. Barnes, Director of our Laboratory, to answer the question propounded by Dr. Fitch as to whether we base our laboratory diagnosis on the finding of negri bodies or inoculation of animals or both.

DR. BARNES: In some instances, the heads are too badly decomposed for satisfactory examination, and in those instances an examination cannot be made, especially in hot weather, but in our preliminary examination if any Negri bodies are found, the case is considered positive. If they are found on sections, the case is considered positive.

Animal inoculations are made only in cases where we have a history that persons or animals were bitten. If we do not have such a history, animals are not inoculated.

DR. ADOLPH EICHHORN: The papers presented on rabies are of the most importance, the one presented by Dr. Munce dealing primarily with the regulatory control of the disease and those presented by Drs. Barnes and Schoening discussing the control of the disease by vaccination.

Dr. Munce, in his very interesting paper, pointed out the possibility of controlling the disease by quarantine measures together with other restrictions with the object of reducing the number of cases of rabies. Similar efforts have been undertaken in many localities throughout the United States, but very little progress has been made in controlling the disease by such procedures. It surely cannot be our aim just to drift and let rabies exist in certain territories so that it persistently menaces the public health.

While Dr. Munce might have been partly successful in his state, the available data does not show any progress towards control in other states. Even the figures presented by Dr. Munce do not indicate that the control measures have

been successful in the state of Pennsylvania, since in his summary he states that in the last two and a half years over 500 positive cases of rabies occurred within the state, and as a result of the occurrence of the disease in dogs, no doubt many hundred persons must have been exposed to the disease.

If we consider this situation with regard to rabies throughout the United States, we, no doubt, would get appalling figures, not only on the occurrence of the disease in dogs and animals, but also with regard to the number of persons who were compelled to take the rabies treatment. I dare say that I would not exaggerate in my estimate by stating that 30,000 to 40,000 persons are required to take the rabies treatment annually in the United States. The goal of the veterinary profession should be towards actual control of rabies with the possible ultimate aim of its eradication, but even with strict quarantine measures and with rigid enforcement of the law, as carried out in Pennsylvania, no progress has been made in that direction.

It is evident that with the present methods it will not be possible to control the disease and much less so to eradicate it. It is my opinion that research work along the lines as reported by Drs. Barnes and Schoening may develop a more workable plan which could be more readily controlled and enforced.

With the development of a successful method of immunization, it would be possible to control the infection, as proven by the results in Japan.

The papers of Drs. Barnes and Schoening on the experimental immunization do not coincide. Dr. Barnes pointed out that phenolized vaccine cannot be depended upon to produce immunity. However, at the same time he pointed out that the dose of infective virus injected has been determined only in a relative way. Dr. Barnes promises continuation of these experiments which might establish or disprove the value of the method.

On the other hand, in the paper presented by Dr. Schoening he points out an effective method of immunization by a vaccine, in which the virus has been treated with chloroform. His data on the method appears to be very conclusive. If the procedure can be depended upon to produce an immunity lasting even a year, it will be possible to recommend the treatment and ultimately aim towards compulsory vaccination.

It is hoped that Dr. Schoening will continue the experimental work and also that others will conduct similar experiments so that if the method is dependable it may be immediately adopted as a standard procedure of vaccination against rabies. No doubt, in developing the chloroform treated rabies vaccine, Major Kelser is deserving of a great deal of credit as he was the first to point out that a chloroform-treated virus will more effectively immunize dogs against rabies than a phenol-treated vaccine.

The charts shown by Dr. Schoening are certainly conclusive proof of successful protective vaccination of rabies. He definitely showed that practically every dog receiving the chloroform treated vaccine proved immune against subsequent virus injections, whereas practically all of the controls died.

While Dr. Schoening considers his work only as preliminary, it is hoped that no time will be lost by him and others to definitely determine the value of the chloroform treated vaccine. If the procedure is effective, the Bureau of Animal Industry should not hesitate to require that all rabies vaccine used for prophylactic purposes be prepared according to such a procedure and, with the backing of the authorities, dependable results may be accomplished by the vaccination throughout the country. (Applause)

DR. MUNCE: I believe that I would be failing in my duty if I were to let go unanswered Dr. Eichhorn's statement that we are not making headway in controlling rabies in Pennsylvania.

With a dog population of 700,000 as we have in Pennsylvania and only 640 cases of rabies in a period of two and one-half years, and in much of the territory covered year after year we have not a single case of rabies reported, I think it is unbecoming the last speaker to say that we are not controlling rabies in Pennsylvania and indicates that he is on unfamiliar ground.

What is meant by eradication? Do you mean permanent eradication? If so, then one might say that we are not eradicating any disease. We are not eradicating tuberculosis. We are not eradicating glanders, or any disease among animals or among human beings. Permanent eradication means permanent—forever.

I do not believe that we have reached that stage. Our records will prove that we are not only controlling rabies by the quarantine method but eradicating outbreaks of this disease in Pennsylvania.

If anyone questions my statements, then I invite him to come to Hatrisburg and we will produce the records to back them up.

DR. EICHHORN: I didn't mean to infer that Pennsylvania is not doing as well or better than any other state in the Union. What I wanted to point out is the fact that in the United States we are not making progress towards controlling rabies, and when I speak of eradication, I mean eradication, because that is one disease that can absolutely be eradicated if concerted action is taken towards that goal.

The problem is not such as we have with a disease like tuberculosis. It is not a disease such as many other contagious diseases we have in the United States, and the fact that Pennsylvania still has, in two and one-half years, over 500 cases of rabies in dogs, I think shows that we are not getting where our ultimate goal should lead us. That is the point I want to bring up. Rabies is an eradicable disease, as proven in many other civilized countries, and that should be the ultimate goal toward which the veterinary profession should work. (Applause)

DR. JOHN REICHEL: I want to refer back for a moment to the technical discussion that we listened to here. I think in previous presentations of research work with rabies, a good many of us here have wondered whether or not there were data to support the contention of dead rabies vaccine being capable of immunization.

We have had presented to us evidence that if all of the materials are handled properly it can be demonstrated that dead rabies vaccine immunizes. I think that is clear cut.

I have always said in previous discussions of this sort, that the infective dose and its method of administration was exceedingly difficult in connection with rabies vaccine experiments. Intramuscular injection seems to be, perhaps, a ittle m ore reliable than the others in connection with these experiments.

Briefly, however, I want to state that in connection with the preparation of rabies vaccine, when a brain is taken from an animal for producing the vaccine, it has been definitely shown, in experimental work, that the virus itself is not uniformly distributed through the brain of the animal. Years ago experiments were conducted to demonstrate the presence of virus in various parts of the brain, and it varied. It also varies with the time of death of the animal. In other words, if an animal with symptoms is allowed to remain alive until the very end, the degree of concentration is greater. In the commercial production of rabies vaccine, it is always customary to harvest rabies vaccine when the animal shows well-pronounced symptoms.

We have heard some reference made to increased doses, from 5 cc to 10 or 20. I don't think it is generally known that there has been considerable variation in the amount of brain material in vaccine, and I don't think we are all informed as to even the basis on which we started work.

We heard the statement made that a calculation is made first of the brain material kept in glycerin for a period, and then so many grams of this glycerinated brain material taken as a basis to start with. It is rather unreliable to start with anything other than the total solid content. That can actually be determined chemically by drying and weighing, and here are some of the factors: Fresh brain material contains 75 parts water. A 20 per cent suspension of brain material actually contains only 5 per cent total solids.

DR. E. B. ACKERMAN: I am rising more for a point of information than to discuss the papers that were presented, but I have two cases in point that I would like to present. One was a little pet dog that had all of the symptoms of dumb rabies. The only person who was bitten was myself, and when we sent the brain to the laboratory, the report came back positive. On the strength of that I took the Pasteur treatment. Some two months later, after they had made animal inoculations, the report came back negative, that the animal had not died of rabies, or shown any rabies.

On the other hand, we had a police dog in our town that had bitten two children, and it showed all of the symptoms of furious rabies. The laboratory findings in this case were negative, and these children were not submitted for Pasteur treatment. About twelve or fourteen days later, they reported that the inoculated animal proved positive.

Now, there are two cases in which the two examinations—the brain examination and the animal inoculation—were contrary, and I would like to know if that happens frequently, or if it should have happened, or whether there was some laboratory error, or what.

DR. REICHEL: It happened to be my lot, from the year 1904 to 1910, to make routine examinations of dogs' brains, and I think we were able to set up in those years that when Negri bodies were actually found, an animal test never failed. Naturally, if brain material comes in, and it isn't in excellent condition, a worker may have difficulty in making up his mind about the presence or absence of Negri bodies, and he may venture to express an opinion which is not confirmed later by analyses. Furthermore, in this particular instance, there may have been some difficulty with the animals. I can't say. But I don't think we need to question any longer the fact that when Negri bodies are present virus is there, and the animals that are injected will come down.

DR. C. H. CASE: I would like to ask Dr. Munce whether every veterinarian has to report every case of rabies that comes to his notice.

DR. MUNCE: Yes, the law requires that all transmissible diseases be reported.

CHAIRMAN KELSER: I just want to say a word in connection with Dr. Schoening's paper in regard to our chloroform-treated vaccine. That work was undertaken in the Philippines, following our rinderpest work, and initially we inoculated only two rabbits subdurally with vaccine prepared according to that method. It so happened that they did not come down with rabies, and we went ahead with our vaccination work, giving the vaccine subcutaneously and had no particular difficulty at that time. We concluded on the basis of the action of chloroform on rinderpest virus and on the basis of these two rabbits we had inoculated subdurally and our subcutaneous vaccination, that our virus was actually killed in a very brief time.

However, after getting back to Washington and continuing our work there, we determined that our first conclusion was wrong; that the virus of rabies was not killed immediately, as was the case with rinderpest, but is killed only after a rather prolonged period.

In that respect our subsequent investigations have followed along the lines of those of Dr. Schoening. We have put on several tests, and since arriving here one of my assistants sent me the conclusion of an experiment to determine whether or not the virus in the last lot of vaccine was killed within a definite time, and this is the conclusion:

"After 11 days we had one rabbit"—that is 11 days after the chloroform had been added to the vaccine—"come down with rabies as a direct result of subdural inoculation of that vaccine."

Prior to that time, putting on two rabbits from 72-hour periods up, we, of course, found that the virus was still active, but in two weeks the virus was rendered inactive by the use of chloroform. In other words, we must save the vaccine approximately two weeks before it is safe to use it, and I think that is what Dr. Schoening found in his work—that approximately two weeks is required before the virus is killed.

DR. REICHEL: Will you state the temperature?

CHAIRMAN KELSER: Ice-box temperature or room temperature, and no incubation at all.

DR. E. M. PICKENS: For the benefit of the record and for personal information as well, I would like to ask Dr. Barnes if in his experiments it is customary to report animal inoculations negative in rabbits at the end of ten days.

DR. BARNES: We do not report our animal inoculations for 100 days. I showed one rabbit there on the chart that did not succumb until 206 days; so I don't see why it should be reported in ten days. We had only one animal in our whole group that died of rabies in 12 days. Twelve days is the shortest

period, except that there was one dog in the group that died in five days following a second exposure. Now, that is along the line of what Dr. Kelser has said.

This virus of which we prepared the emulsion on November 20 stood in the refrigerator at ice-box temperature until February 14 when we inoculated four dogs with two different strains of virus, two with each strain, and on May 14 we again exposed those dogs to another strain of virus, and the four died of rabies following that exposure. Two died in 18 days, one in 19 days, and one in 5 days. The one that died in five days showed symptoms two days following the inoculation. That may possibly have been due to the virus that was inoculated on February 14, which was kept in the refrigerator from November 20.

Imported Milk Now up to High Standard

As a result of strict enforcement of the federal Import Milk Act, a much smaller quantity of milk was imported into the United States during 1929 than in the year before, and the general level of quality of the milk imported in 1929 was much higher, according to H. B. Switzer, chief of the Rouses Point (N. Y.) inspection station of the Food, Drug and Insecticide Administration. The act was passed February 15, 1927, but the work of complete enforcement of it was not begun until June 1, 1928.

Sour milk and cream are not allowed to enter the country, on account of high bacterial count; only sweet milk and cream which meet the strict requirements of the law may come in. All products coming within the jurisdiction of the act are now being pasteurized, either before or after importation, and the American consumer of imported miik and cream may rest assured that the products are safe, clean and of high quality.

Since June 1, 1928, the number of permits held by importers of milk and cream decreased 50 per cent. The Canadian shippers under Rouses Point inspection, from whom most of the imported milk is received, held a total of 945 permits on June 1, 1928. Since that date 459 permits have been cancelled, leaving 486 in effect on October 1, 1929.

Cancellations of dairymen's permits were due to failure to identify shipments properly with tags; offering for entry milk or cream higher in temperature than 50 degrees F.; shipping milk that exceeded the permitted bacteria standards; and failure of dairies to pass inspection of field veterinarians.

Permits of pasteurization plants have been cancelled for receiving milk or cream from unapproved sources, failure to pasteurize properly or to keep accurate pasteurization records, using contaminated water, and failure to maintain proper sanitary conditions of the plant or equipment.

THE DEPARTMENT OF ANIMAL PATHOLOGY AT CAMBRIDGE UNIVERSITY*

By E. L. STUBBS, *Philadelphia, Pa.*

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During the summer of 1928, the writer had the opportunity of visiting the Department of Animal Pathology at Cambridge University, Cambridge, England. This Department is now six years old and has been organized for the purpose of conducting research in the diseases of farm animals. It has been founded along lines which appear admirable for this purpose and which seem to be fundamentally important in organizing such work. This institution has arisen as a result of the desire of the British Government to provide facilities for investigating live stock diseases.

The Ministry of Agriculture and Fisheries devised a plan for the establishment of a professorship of Animal Pathology in a university. It was located at Cambridge, because of the presence of a strong medical center and because Cambridge is the center of a rich agricultural tract with much live stock. The Ministry of Agriculture and Fisheries granted funds, which were turned over to the University to provide income on the condition that such income would be used to found a chair of Animal Pathology, to pay the salary of a professor and to defray other expenses in connection with the work of the chair. The University accordingly created a chair of Animal Pathology, to which Professor J. B. Buxton, F. R. C. V. S., was elected in August, 1923, and he devised the scheme of development and is directing the work of the Department.

Permanent quarters are being provided in the new pathology building at Cambridge University. This new building is a fine, four-story, brick structure and is located near Professor Nuttal's biology laboratory which is known all over the world. The new pathology building has about one hundred rooms, the construction of which is being aided by Rockefeller Foundation funds. The first story is to be used for administration and service, the second story for teaching, the third story for research and the fourth for experiment animals. Some of the interesting features

*Presented at the sixty-sixth annual meeting of the American Veterinary Medical Association, Detroit, Mich., August 13-16, 1929.

in this building are cork and wood-block floors, and windows which turn in and can be cleaned from the inside of the building. All drain-boards are located above sinks to conserve space. The building provides for the teaching of the fundamentals of path-

FIG. 1. (Above) Field Station—Department of Animal Pathology, Cambridge University. (Middle) Private Laboratory. (Below) Stables.

ology to medical students as well as providing a special course in pathology. This instruction is given in connection with a six-year course in medicine. The Department of Animal Pathology

is being provided in the new building with six laboratories and two offices, together with the various services in common with human pathology, as technical assistance in histology, histopathology and culture media preparation, museum, lecture-rooms and library facilities.

The present organization consists of the Professor of Animal Pathology in the University of Cambridge, who is the Director, an Assistant Director, who is a pathologist, four other pathologists and two biochemists, with four laboratory assistants and two animal attendants.

Field laboratories are also maintained on Milton Road, in Cambridge, consisting of four laboratories and two cottages, with several sets of stables and pens for animals. The work is all being done in these quarters at present.

The Department has been formed for investigations into the diseases of farm animals. Members of the staff are now enengaged in special research. A branch has also been created to care for routine diagnosis of pathological material sent in by veterinarians, control officers and agricultural organizers. This keeps the Department informed regarding serious outbreaks of disease, indicating problems, as well as furnishing material for investigation.

The Department has also organized a mobile section facilitating the carrying out of investigations in the field and on premises where outbreaks of disease are occurring. It consists of a motor laboratory built and equipped so that it has a wide range and is provided with ample facilities for carrying out any laboratory investigation and even for prolonged field investigations. The motor laboratory is a truck-like affair fitted up as a laboratory.

Mary problems are being undertaken by this Institute of Animal Pathology among which are the following: tuberculin tests, the potency of tuberculin, swine tuberculosis, tuberculosis protective vaccination, filtrability of tubercle bacilli, sheep diseases, contagious abortion of ewes, mastitis in ewes, joint-ill or polyarthritis of lambs, pustular stomatitis, parasitic gastroenteritis and diseases of swine.

It seems that the arrangement is ideal for research work. Liberal provision of funds from public money is provided through the University, yet the administration is free and unhampered. Members of the staff engaged in special research are available to form a team for special investigations. It is felt that such a hook-up provides the valuable facilities of a great university in

the way of furnishing advice on special problems, library, museum and service facilities, as well as close cooperation with workers in other departments. The close association with workers in allied sciences, particularly the medical group, is broadening and inspiring. Such an atmosphere contributes to good, intensive research work of the highest order.

Substandard Ether Seized

The largest shipment of ether for anesthesia ever detained by the federal government was seized at Bayway, N. J., recently, after laboratory tests had shown that samples from a lot consisting of 108,300 quarter-pound tins were below the standards required under the federal Food and Drugs Act. The seizure was made by the Food, Drug and Insecticide Administration, U. S. Department of Agriculture, which enforces the Food and Drugs Act. This ether did not meet the requirements of the U. S. Pharmacopoeia, which is the standard designated by the Food and Drugs Act for drugs in interstate commerce or imported from abroad.

The seized ether is part of a lot made for the government during the World War and kept in storage until 1926, by which time it had deteriorated to such a degree that it was unfit for use as an anesthetic. The War Department then sold it at a low price, under bond that it was not to be used or resold for use as an anesthetic but only for technical purposes, such as in laboratories, for dry cleaning, or for fuel in starting motors. Contrary to the terms of the bond, some of this ether, labeled as anesthetic ether, was consigned to hospitals in small lots. These small lots were seized at once. Now this large shipment has been removed from the channels of trade by action under the Food and Drugs Act.

Although improvement has been made in the manufacture and packaging of ether in the last few years, some ether still shows deterioration upon standing in sealed tins. For this reason authorities have been especially vigilant in the inspection of ether. Whether or not ether that has deteriorated is harmful to patients on the operating-table, a matter upon which medical authorities do not entirely agree, is quite beside the point in the administration of the Food and Drugs Act. It is the duty of the officials enforcing this law to remove from interstate commerce all ether that fails to meet the standards set by the U. S. Pharmacopoeia.

AN EXPERIMENTAL STUDY OF DRUGS STIMULATING THE MOTILITY OF THE RUMINANT STOMACH*

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Drugs having a stimulant action upon the muscular tissue of the ruminant stomach and, by virtue of this action, aiding in restoring depressed states of rumination to normal, are designated as ruminatorics.

They rank high among the important drug classes of veterinary medicine and in addition have the unique distinction of being one of the most therapeutically restricted drug classes existing within the realm of pharmacology, their application being confined exclusively to the ruminant species of animals. They are of pharmacologic and therapeutic interest only to members of the veterinary profession.

Experimental veterinary pharmacology has progressed very slowly in past years and the present day finds but few workers engaged in this extensive, interesting, and extremely important field of study. One of the reasons for this neglect is the lack of adequate physiological data pertaining to many of the domestic animal species, such data constituting an absolutely essential prerequisite to any pharmacological study.

Physiological details concerning the process of rumination have not been available until rather recent years and in consequence an experimental study of the effects exerted by drugs upon this particular function has remained an impossibility.

In 1921, the writer was afforded the opportunity of collaborating in an experimental survey of gastric motility as exhibited by the bovine stomach and also the phenomena of rumination. This project extended over an interval of four years and resulted in an accumulation of technical and physiological data which forms the essential back-ground for the experimental work to be reported in this paper.

The particular objective of this work has consisted: (a) of determining the relative efficiency of the various drugs which are considered by clinicians to possess ruminatoric properties and (b) of determining the effects exercised by each drug when given in varying doses.

*Presented at the sixty-sixth annual meeting of the American Veterinary Medical Association, Detroit, Mich., August 13-16, 1929.

Method of Study

Graphic records were obtained from the rumen and reticular cavities and conclusions were drawn from a comparison of the records previous to and after the drug administration. The recording was accomplished by means of small rubber balloons located in the near vicinity of the stomach walls and arranged so that the contraction movements of the walls would exert a compression effect upon the partially inflated balloon. Each balloon was connected by suitable rubber tubing to water or, better, to saturated magnesium sulphate manometers, which recorded upon the moving carbonized surface of a kymograph drum. Permanent rumen fistulae, of sufficient size to admit the recoring apparatus into the stomach interior or the observer's hand and arm, were established in the experiment cattle. The balloons were inserted through the fistula and held immobilized in any desired position by means of heavy wire supports. Displacement of balloons by the moving food mass has proved to be a serious problem, as a slight change in location causes such pronounced changes in the record as to render worthless the results of many experiments.

Constant, unvarying conditions were maintained to the greatest possible extent throughout each experiment, a requirement which has made the work extremely exacting.

Each graphic record obtained contains simultaneous tracings of rumen and reticular contractions combined with a time tracing.

Physiological Review

Some of the functional phenomena of the rumino-reticular portion of the bovine stomach will be reviewed briefly at this time in the interest of a more comprehensive understanding of the drug actions discussed later.

Food, during the course of its passage through the first three compartments of the ruminant stomach, undergoes preliminary changes that are of vital importance to the digestion process as a whole. The changes effected in the nutriment are chiefly physical in nature, the food materials being softened and finely subdivided. Important chemical disintegrations also are completed in this portion of the alimentary canal by bacterial action. These processes are all dependent upon a generous supply of moisture, such as is provided by ingested water and swallowed saliva, and upon an active state of gastric motility. This motility brings

about an intimate mixing of the liquids and solids in the stomach and supplies the means for transporting the food along its normal course through the stomach. The nonstriated muscle of the stomach walls contracts in rhythmical manner, the contractions appearing first in the depths of the reticular cavity from which point they travel as peristaltic waves in a posterior direction over the walls of the rumen. Nonstriated or visceral muscle has certain functional characteristics which require mention in connection with this particular discussion. The property of automatic functioning is well developed in this tissue, the term automatic referring to the ability of a tissue to function in the absence of any nerve control. Nerves are supplied, however, to the gastric muscle for the purpose of varying the functional activity of the tissue in accordance with the demands of the body for food material. Two sets of nerve fibers regulate the gastric motility. One set, contained in the vagus nerve trunks, carries motor nerve impulses and thus serves to increase the activity of the stomach walls; the second set, supplied by the splanchnic nerve trunks, carries impulses that depress the gastric motility. These two sets of nerves are constantly exercising their control over the muscular tissue of the stomach, this control being reflex in manner of operation, the sensory part of the reflex arc consisting of nerve fibers that carry sensation of taste, pain, etc. Some of these sensory nerves are distributed to various portions of the gastro-intestinal tract.

Under normal conditions a state of constant motility is maintained by the stomach walls, one part contracting, while another relaxes and rests for a brief interval, and vice versa. In the rumen and reticulum large quantities of solid and liquid ingesta a retemporarily stored and it is in this portion of the stomach that the food is prepared for rumination. The bolus of rumination or the "cud" consists of material derived from the reticulum; a carefully timed series of muscular contractions, executed (by the reticular walls, diaphragm, and esophagus) in the order named, transfers the material to the mouth in quantities convenient for mastication. The fact that a considerable number of muscles are involved and that strict co-ordination of contraction is required is evidence in favor of the existence of a controlling nerve center, the rumination center, which brings into harmony the various muscular reactions concerned in rumination. Following the second mastication or rumination, the food material is returned once more to the anterior part of the rumen cavity.

Recent investigational study has revealed the fact that the esophageal groove does not function as a guiding medium for food and drink in the mature ox; this structure does function, however, in the nursing calf, serving to guide the milk diet of the young animal into the fourth compartment or abomasum. This resumé, dealing with the functional phenomena of the rumen and reticulum, has made mention of the vital importance of moisture and motility. In the partial or complete absence of either or both of these factors, rumino-reticular digestion fails and the conditions of atony and impaction become established.

In the light of these physiological facts it appears that rational treatment should deal first with the matter of correcting any possible deficiency of moisture which may exist; with this accomplished, the next phase of the treatment logically consists of applying stimulation to the partially or completely depressed neuro-muscular tissues of the stomach walls. Such stimulation is most conveniently accomplished by means of drug action. In this study, as far as it has progressed, the most representative of such drugs have been selected for experimental observation, their individual actions being determined and no combinations having been used.

The list consists of:

1. Tartar emetic
2. Barium chlorid
3. Arecolin hydrobromid
4. Pilocarpin hydrochlorid
5. Eserin salicylate
6. Lobelin sulfate.

The first two drugs mentioned in the list have enjoyed great popularity in recent years, being used singly and in combination. Lobelin sulfate is a late addition to the list.

Tartar Emetic

This double salt of antimony disintegrates when taken into the stomach and the irritant action of the liberated antimony ion causes emetic effects in animals capable of vomiting. Vomition is not commonly observed in ruminant animals and a nervous mechanism for the control of such an act is not considered to be present. Under these conditions the antimony irritation affecting the gastric mucosa, although failing to cause vomiting does reflexly stimulate gastric motility and upon this action is founded the use of the drug for ruminatoric purposes.

In our experiments tartar emetic has been used in doses ranging from 15 to 150 grains. These administrations were given as drenches in one-half to two pints of water and also as the undissolved drug in gelatin capsules. The methods of administration consisted of drenching and of inserting the drug through the fistula, usually into the reticular cavity. In addition, the drug has been applied in one per cent solution to the rumen and the reticular walls, and by means of tubing has been injected directly into the abomasal compartment.

When given by mouth or by way of the fistula into the rumino-reticular cavity the effects of 60-grain doses have not been especially pronounced. The great quantity of moisture present and the active motility of the normal rumen and reticulum apparently serve to diminish the intensity of the drug action by extensive dilution and by retarding the progress of the drug into the abomasum.

The drug in 120-grain doses per os has been observed to produce a very pronounced increase in the strength of rumen contractions, this action becoming apparent one and a half hours after administration. The improvement continued for two hours and was still fully developed when recording was suspended. Tartar emetic per os appears to exercise its effects chiefly upon the rumen muscularis. Thirty grains in two ounces of water, introduced directly into the abomasal cavity, is followed in about one-half hour by an excellent stimulant action which affects the strength of both rumen and reticulum. Coughing and labored breathing have frequently been observed to precede the period of stimulation, these symptoms developing within a few minutes and possibly being due to reflex stimulation of the respiratory center. With sixty grains toxic symptoms appear within forty to fifty minutes, the pulse becoming very weak, with cold extremities and skin surface in general; an exceedingly strong and persistent depression of rumen and reticular movements occurs which has been recorded for four and five hours after its appearance and remained still fully developed at the end of the recording period.

Tartar emetic stimulation of rumino-reticular motility does not appear to be the result of irritant action upon the rumen or reticular mucosa, as a long latent period precedes the appearance of any effects after administration by way of the mouth. This period of latency may be considered to be the result of a delayed passage of the drug into the abomasum where the reflex action is

initiated. To test out the possibility of tartar emetic reflexly stimulating motility by action upon the rumino-reticular mucosa, a one per cent solution of the drug was applied by means of a cotton pledget to portions of the rumen and reticular mucosa. The only effect observed was that of decreasing the frequency of the rumen and reticular contractions for a period of several minutes. Ninety-five per cent alcohol pledgets applied to the rumino-reticular mucosa caused complete suppression of the reticular movements and reduced the frequency and force of the rumen contractions, the symptoms being quickly relieved by the application of water to the alcohol-treated area. These results demonstrate quite clearly that irritation of the rumino-reticular mucosa is productive of depression rather than stimulation of motility.

Tartar emetic may be considered as one of the strong ruminatorics when used in doses of 90 to 150 grains. It will be favored in its action by generous dilution at the time of administration, which should reduce the degree of irritation within the reticulum, and facilitate passage of the drug solution into the abomasum.

Barium Chlorid

This drug in sufficient concentration exercises an irritant action upon the mucosa of the gastro-intestinal tract and thus reflexly stimulates the muscle tissue of this region. Following absorption the barium ion has a direct stimulant effect upon all varieties of muscle tissue.

In our experiments with barium chlorid the same methods of administration were used as described in connection with the work on tartar emetic. The dosage was the same except that the maximum oral dose of the barium salt was increased thirty grains.

It may be stated in brief that the results of these experiments have failed to give any evidence that stimulation of the rumino-reticular muscle tissue occurs with the dosage usually recommended in practice. Quantities ranging from 60 to 180 grains have been administered per os and the motility recorded for periods of four and five hours following the administration. No change could be detected in the rhythm, frequency or strength of contractions. Further study of this drug in heavier dosage is planned for future consideration. Thirty grains of barium chlorid in two ounces of water injected directly into the abomasum have produced a slight increase in reticular strength, this action appearing one and a half hours after administration; no

effects upon the rumen. Direct application of two per cent solution of the drug to the rumen mucosa was without any effect; applied to the reticular mucosa, slowing of the reticular movements occurred with a very transient increase in the strength of rumen motility.

In this work with barium chlorid we have not observed any action with the therapeutic dosage used that would justify a classification of the drug among the ruminatorics.

Arecolin Hydrobromid

Arecolin has a stimulating action upon certain nerve terminals and other structures belonging chiefly to the parasympathetic portion of the autonomic nervous system. As the parasympathetic innervation of the stomach is concerned with the bringing of motor impulses to the muscle fibers, it must logically be concluded that arecolin acts as a stimulant of gastric motility.

With the above pharmacological data in mind, we were surprised to note that half-grain doses of arecolin, used in the initial experiments, caused complete depression of rumino-reticular movements for intervals ranging from forty to fifty minutes, with no apparent improvement in motility succeeding this action.

In further experiments with reduced doses of the drug, it was discovered that no depression appeared with administrations less than one-fourth grain, and that very pronounced stimulant action resulted from doses as small as one-sixteenth grain. One-thirty-second grain gave no apparent results. Doses of one-sixteenth and one-eighth grain were found to be especially efficient, the improvement in the strength of the rumen and reticular contractions appearing in five to twelve minutes and persisting for one and one-half hours. The reticulum is more strongly affected than the rumen. These small doses act without causing any evidence of abdominal pain or other disturbance. One-half grain causes severe distress, due to the intestinal spasm combined with some circulatory and respiratory disturbance. From our observation there appears to be a close relationship between the intensity of abdominal pain and the degree and duration of gastric depression. As it is a well established fact that the stomach may suffer reflex depression due to sensory disturbances occurring in any portion of the body, it appears quite logical to attribute arecolin depression to reflex pain inhibition. Other factors may, however, be concerned which are less apparent at this time.

These experimental results indicate that arecolin is not being used to advantage as a ruminatoric at the present time, the drug being commonly employed in one-half or even one-grain doses. Such administrations are unsafe and not infrequently cause sudden death due to cardiac failure. Doses of one-sixteenth and one-eighth grain are well within the safety zone and have a powerful stimulant action upon the walls of both the rumen and the reticulum.

Pilocarpin Hydrochlorid

The pharmacological action of pilocarpin resembles very closely that of arecolin, but the drug is much less powerful in its effects.

Three-fourths and one-grain administrations of pilocarpin increase the strength of the reticular, and to a lesser extent the rumen movements. The fact that this action is not pronounced and is brief in duration classes pilocarpin among the weak ruminatorics. Similar to arecolin an inhibition of motility is produced by relatively large doses. A dose of one and one-half grains completely checks reticular contractions for fifteen to thirty minutes and slows but fails to check the contractions of the rumen. If used as a ruminatoric, one-grain doses should be administered.

Eserin Salicylate

One of the important physiological actions attributed to this drug is that of increasing the motility of the gastro-intestinal tract. The manner in which the drug accomplishes this particular action is not definitely understood at present, the peripheral effect being exerted either upon the parasympathetic motor nerve terminals or upon the muscle fibers directly.

The results obtained with arecolin greatly aided the progress of the work upon eserin. Starting with one-eighth grain, the size of the dose was increased to one-fourth, one-half, three-fourths and one grain. No action was obtained with one-eighth or one-fourth grain. One-half grain improves the strength of the reticular contractions within a few minutes. The strength of the rumen is also increased but does not usually appear as promptly as the reticular action. With three-fourths grain a very pronounced stimulant reaction occurs, the rumen and reticular movements becoming extremely powerful, this reaction remaining established for over two hours. The animals did not exhibit any symptoms indicative of abdominal pain until the one-grain dose was tried. This dosage, although productive of severe

distress, failed to bring about a cessation of motility as occurred in arecolin action, a fact which indicates that some marked difference exists in the gastric action of the two drugs. The failure of the well-marked pain reaction to check motility reflexly points quite strongly to the drug having a direct stimulant action upon the gastric muscle, which causes this tissue to remain unaffected by any inhibitory nerve control.

Eserin action has stood out with particular prominence among the various drugs experimented with in this study and it approaches the requirements of the ideal ruminatoric more closely than any drug studied thus far. The therapeutic dosage should not exceed three-fourths grain in single doses, which may be repeated in one hour to advantage.

Lobelin Sulfate

Lobelin resembles the alkaloid nicotin in exerting a stimulant and later a depressant action upon autonomic ganglion cells in general, both sympathetic and parasympathetic. This indiscriminate type of action greatly complicates the pharmacology of this drug.

Lobelin sulfate used in doses of one-tenth, two-tenths, one-quarter and one-half grain has been observed to improve the strength of the rumen and reticular contractions. This stimulant action is much less powerful than that obtained with eserin and arecolin salts. The fact also has been noted that in ascending therapeutic doses the action fails to increase in accordance. Repeated experiments have demonstrated that a rather wide range of action is obtained with the same dosage in the same animal. It may be possible that the potency of the drug differs in the various samples of hypodermic tablets or the explanation may involve the activity of the endocrine tissues, the secretions of which are closely associated with the functions of the autonomic nervous system.

Repeated administrations of lobelin at short intervals have revealed the fact that the drug may cause depression of ruminoreticular motility in total dosage varying from eight-tenths grain in some experiments to two and seven-tenths grains in others. These results indicate quite clearly that certain factors are modifying the drug action to a very unsatisfactory degree. It is difficult to define the particular range of dosage productive of the most efficient stimulant action, but any amount up to three-fourths of a grain may be considered free of depressant effects.

SUMMARY

1. Certain drugs commonly used as ruminatorics have been studied experimentally to determine the degree of any stimulant action upon the ruminoreticular muscularis. Observations were made by means of graphic records of ruminoreticular motility.

2. Drug stimulation affected principally the strength rather than the frequency of the gastric movements.

3. Tartar emetic (120-150 grains), well diluted with water, gave excellent results.

4. Barium chlorid is apparently of little value as a ruminatoric.

5. Eserin salicylate in doses of one-half and three-fourths grain produced the most powerful motility observed during this study. Two administrations one hour apart were found to be particularly efficient.

6. Arecolin hydrobromid was very efficient in doses of one-sixteenth and one-eighth grain. Administrations of one-fourth grain and above cause depression of gastric motility for three-quarters to one hour.

7. Pilocarpin hydrochlorid failed to exhibit more than mild stimulation in one-grain doses. Depression appears with one and one-half grains.

8. Lobelin sulfate was found to be quite variable in action and with pilocarpin may be classed as one of the weaker ruminatorics.

STATE BOARD EXAMINATIONS

Iowa Veterinary Medical Examining Board. State House, Des Moines, Iowa. January 8-9, 1930. Dr. Peter Malcolm, Division of Animal Industry, State House, Des Moines, Iowa.

California State Board of Examiners in Veterinary Medicine, Davis, Calif. January 7-8, 1930. Dr. G. E. Middlehoff. Secretary, Oroville, Calif.

Silver Anniversary

In renewing his advertising contract for 1930, Mr. F. V. Kniest, of Omaha, Nebr., directed attention to the fact that he recently celebrated his silver anniversary in business, having started in 1904. Mr. Kniest is proud of the fact that he can count on the fingers of one hand the complaints received from his patrons during this time. The fact that he does business in forty-eight states "under the mails" is pretty good evidence that he is on the square.

DISEASES AND PARASITES OF SILVER FOXES AND APPROPRIATE TREATMENTS*

By J. E. SHILLINGER, *Washington, D. C.*

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A growing need for satisfactory methods of maintaining the health of foxes has been brought about by the rapid increase in the number of these animals kept in captivity on fur farms for commercial production of fur. If the desired information is not available from local veterinarians, it will be sought among the laity. In every community you will always find one or more poorly informed individuals ever willing to furnish treatments for "curing" every malady to which these animals are subject. The damage that is done by these treatments is closely seconded by the patent nostrums sold under misleading advertisements claiming phenomenal results.

These conditions are in no particular sense different from those encountered among the breeders of domestic live stock. But since, however, many veterinarians are unfamiliar with the anatomy, food habits, and general characteristics of those animals usually raised and kept for furs, they frequently avoid giving their professional attention and thus encourage the excuse for unscientific treatments.

The most prominent fur animal kept solely for its pelt is the silver fox. It is a common misconception that the fox and dog are so closely related that they are subject to the same parasites and diseases and respond to the same treatment. The purpose of this paper is to attempt to draw some of the more obvious comparisons of the ailments to which these animals are subject and to outline control measures.

DISTEMPER

Fox ranchers as well as some veterinarians usually refer to every disease in foxes for which no apparent cause is known as "distemper." Many of these diagnoses are erroneous, or else distemper in foxes must be defined as referring to a group of diseases showing diverse symptoms, affecting different types of animals, attacking different tissues, and caused by different forms of

*Presented at the meeting of the Montana State Veterinary Medical Association, Missoula, Mont., July 8-9, 1929.

organisms. There are several acute infectious diseases of foxes, none of which present the same symptoms as distemper in dogs.

A group of investigators at the University of Minnesota began work in January, 1925, on two of the more frequently encountered diseases, namely, "fox paratyphoid" and "fox encephalitis." The former of these was especially prevalent a few years ago in widely separated parts of the country, but it has tended to disappear since 1925 and 1926. This may be due to improved sanitary practices on fox ranches resulting from the lessons learned through extensive losses. When this disease appeared on a ranch, the losses were frequently as high as 50 per cent, and as a result some fox farmers were forced out of business.

Fox paratyphoid is an infection especially of young animals. The course of the disease appears to be of considerable duration in the individuals affected, and a pronounced loss of weight is produced. In controlled experimental infections and in epizootics occurring in summer, no purulent discharge from either the eyes or the nose was noted. This is usually a symptom, however, in epizootics occurring in winter months, and at times there is an involvement of the entire respiratory tract. Preceding death the animal shows marked weakness, sunken eyes, and occasional convulsions. Postmortem the intestinal tract frequently shows marked inflammation and the spleen is almost invariably much enlarged and dark.

Paratyphoid organisms can often be isolated from the spleen in pure cultures and the disease reproduced in test animals. Vaccination with a product prepared from the specific organisms causing the disease appears to be effective in controlling these outbreaks. There may be several types of the paratyphoid bacteria affecting foxes.

Fox encephalitis is a disease believed to be due to a filtrable virus, affecting especially the central nervous system. Death is sudden, and the animals show no loss in weight or other symptoms except those of a nervous character. These may be lethargy, hyperexcitability, paralysis, or convulsions. In most instances no symptoms are seen, the animals being found dead. This disease is more virulent for young foxes than for adults, and losses in epizootics vary from 5 to 40 per cent of the ranch population. Outbreaks are especially common when many animals are brought in close contact with one another, as when they are herded together in the fur range or exhibited at a show.

Postmortem examinations of foxes killed by encephalitis are not apt to reveal signs of anatomical changes. The organs *may* appear perfectly normal. In most cases, however, hemorrhages may occur in the pancreas, stomach, intestine, and the thymus, adrenal, or submaxillary glands. Sometimes gross hemorrhages are seen in the brain and spinal cord. The changes regularly found in the brain and spinal cord explain the nervous symptoms accompanying this disease. A perivascular infiltration of cells around the blood-vessels, clumps of round cells, and hemorrhages in the nervous tissue are seen.

Attempts have been made at producing an artificial immunity from this disease, and a fair degree of success can be attained under favorable conditions. Nothing can be advised by way of control measures at present other than the enforcement of the most rigid quarantine.

The two diseases described appear to have been the most characteristic infections found in foxes in captivity, but it is not believed that they are the only diseases to which these animals are subject. Good evidence is at hand indicating that several other distinct ailments occur in epizootic form on fox ranches. The cooperative research organization composed of workers from the University of Minnesota and the Bureau of Biological Survey, United States Department of Agriculture, are extending their efforts in finding means of preventing losses from the various infectious diseases of foxes.

External Parasites

Doubtless one of the most common forms of ailments to which dogs are subject is mange, especially the sarcoptic variety. Sarcoptic mange is not common among foxes, probably because of the system by which they are kept. However, once the mite responsible for this disease gains a foothold on a ranch, one can expect considerable loss. Difficulties are encountered when one resorts to oleaginous preparations. Powders have not been found to be particularly efficacious, and if the disease is not promptly stopped, it is likely to spread to the other animals on the ranch.

Because of the heavy fur-coat of foxes and the nature of the animal, they do not tolerate immersion in dips very well. This method, however, is doubtless the most efficacious that can be applied. Soluble lime-and-sulphur dip, as used in the treatment of sarcoptic mange in dogs, is preferable. The long, dense fur of

foxes frequently allows the disease to reach a relatively advanced stage before it is detected.

Follicular mange is of rare occurrence in foxes, but ear mange, caused by the mite, *Otodectes cynotes*, is common to a large portion of fox ranches. This mite spreads rapidly among foxes, and unless it is kept in check or eradicated, severe losses result from emaciation, inferior quality of fur, breeding disturbances, and even deaths. Ear mange is not difficult to treat. The application of one of the following preparations may be relied upon to control the infestation: Glycerin 95 parts and phenol 5 parts; or ether 1 part, iodoform 10 parts, and olive oil 25 parts; or olive oil 3 parts and kerosene 1 part. These should be used twice, at an interval of a week to ten days. If all the foxes on the ranch undergo treatment at the same time, with two thorough applications of an effective ear-mange remedy, the disease will usually be eradicated from the ranch. The only practical way of treating the disease is to eradicate it.

Under certain conditions foxes are sometimes as heavily parasitized with fleas as are dogs. Since it is inadvisable to dip foxes if it can be avoided, powdered preparations such as pyrethrum, derris and naphthalene are often used. Fairly satisfactory results can be obtained with these substances if persistently used in conjunction with a thorough cleansing and disinfection of the kennels to destroy the eggs and larvae that might be present. Similar methods may also apply in treatments for lice.

Internal Parasites

Foxes are frequently the host of one species of ascarid, the *Belascaris marginata*, which also affects dogs. These regularly become so plentiful on fox ranches that it is the usual practice of fox breeders to begin the routine treatment of all pups for these worms at 3 or 4 weeks of age. The use of a mixture of 1 part of oil of chenopodium in 20 parts of castor oil, at a dose rate of one cubic centimeter for each pound of body weight, is an effective remedy. The average dose for pups 3 or 4 weeks old is about 0.75 cc.

The hookworm most commonly found in foxes, *Unicinaria stenocephala*, is seldom seen in dogs or cats in this country. It is removed with tetrachlorethylene in doses comparable with those used for dogs of the same weight.

Under ranch conditions foxes are seldom parasitized with tapeworms and practically never with whipworms. Frequently the

bladder and sometimes the pelvis of the kidney contain small threadlike nematodes, the *Capillaria plica*. The pathological changes caused by them is not extensive and no treatment for them is recognized.

The nematodes parasitic in the lungs of foxes present a most difficult problem. Two species are recognized, the *Capillaria aerophila* and the *Crenosoma semiarmatum*. Under natural conditions these worms very seldom affect dogs. The eggs of these parasites are exceedingly tenacious in the soil, and pens appear to remain infective many months after the removal of affected animals. A few worms may be present in the lungs and trachea without causing any apparent symptoms. Increased numbers or other suitable conditions may result in the collection of an excess of mucus in the lower respiratory system, which in turn causes a wheezing or rattling sound when the animal breathes. This is frequently interrupted by a hoarse cough. All these symptoms are made more prominent by exercising the affected animal. At times the mucus may become very profuse and may be prominently in evidence at the nares and mouth. As the case develops pneumonia sometimes becomes a part of the picture.

Medication for destruction of the worms is of no recognized value; however, expectorants may aid in relieving symptoms. Most of the adult parasites soon migrate to the trachea from their immature habitat in the lung tissue. They can be removed from the trachea by means of a specially designed brush, which in the hands of a skilled operator may be the instrument for saving many valuable animals. It is frequently necessary to repeat the process several times at intervals of one to three weeks, but if it is carefully done, and if the animals are removed from the source of contamination, complete recoveries often result.

If animals when first showing symptoms of this form of parasitism are placed in pens so constructed as to prevent reinfestation, they usually recover eventually with no other treatment. Pens constructed with a floor made of 1-inch-mesh wire placed on frames about 18 inches above ground are especially effective in controlling lungworm trouble in foxes. The practices for lungworm control giving best results at the U. S. Fur-Animal Experiment Station at Saratoga Springs, N. Y., are described by Dr. Karl B. Hanson, in a mimeographed leaflet of the Bureau of Biological Survey.

SUMMARY

The definite information available thus far indicates that the only disease conditions in foxes which may be treated practically the same as in dogs are certain parasitic infestations. Extreme care must be exercised in the external treatment of foxes by dipping them in fluid preparations. They may suffer from the shock alone as well as from the action of the drugs on their pelts and by a rather free absorption of the drugs through the skin.

While the effect of certain anthelmintics for the removal of ascarids and hookworms is similar to that obtained in dogs, it must be borne in mind that their reaction to the drugs may be very different. The highly sensitive nature of their nervous systems has a profound influence on their physical response to environment and especially to unusual occurrences. The very fact that they so violently resent restraint is an important factor in putting them in an abnormal state during the administration of any treatment. Their temperature, pulse and respiration have very little diagnostic value, and in every way they are in a distinctly different class of animals from those which the average veterinarian is accustomed to handle.

Canned Chicken Becomes Popular

Increase in consumer demand for canned chicken is resulting in greater demand for government inspection of poultry at canning-plants. Inspection for condition and wholesomeness, under the Food Products Inspection Law, is now maintained at eleven poultry-canning plants, by the Bureau of Agricultural Economics, U. S. Department of Agriculture.

This service is rendered by the Department of Agriculture at the request of the plants engaged in canning chicken, and through cooperation with the New York Live Poultry Commission Merchants Association, the National Poultry, Butter and Egg Association, and the Minnesota State Department of Agriculture. The individual birds are examined by a qualified veterinarian at the time the entrails are removed, and birds which are diseased, unwholesome, or otherwise unfit for food are destroyed.

Poultry-canning firms using the service are permitted to state on the labels of their cans: "Inspected and certified by the Bureau of Agricultural Economics, U. S. Department of Agriculture." In September nearly 2,000,000 pounds of poultry underwent inspection at the eleven plants.

BLACKHEAD IN TURKEYS*

By L. E. STARR, *Blacksburg, Va.*
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Blackhead has long been known to occur in every section of the country where turkeys are raised, and is responsible for severe losses. Observations made indicate that the disease is not prevalent to a great extent in flocks that run on well-drained land. It is believed that blackhead as a serious disease will be found to be localized to a considerable degree in those sections where the land is low or on land on which chickens and turkeys are raised in confinement year after year.

The disease is confined almost entirely to turkeys, although occasionally chickens are affected. On some farms it has become practically impossible to raise turkeys by the old methods, almost 100 per cent of the young poults dying before reaching maturity. It is a disease of young turkeys, from three to four weeks of age, until they are almost mature. Old turkeys rarely are affected.

There are two factors involved in the disease, one the blackhead parasite, a microscopic organism which enters the tissues and produces the characteristic changes, and the other the cecal worm which enters the tissues and presumably opens up a way for the blackhead parasite to enter. It is possible that there are other factors that may take the place of the cecal worm in the production of blackhead, but none have been found to date. With our present knowledge of the disease, turkeys do not become infected unless they run on soil contaminated with the blackhead parasites and the cecal worm eggs.

The disease owes its name to the fact that oftentimes birds' heads turn brown when sick. The name, however, is a misnomer. The head of a turkey with blackhead never becomes black, but in many cases has a somewhat bluish color. This condition occurs in other diseases and is due to disturbances in circulation. A period of ten days or two weeks usually elapses between the beginning of the disease until the bird first shows symptoms of being sick. In exceptional cases young birds only a few weeks old may die suddenly without showing any previous indications of being sick. These are rare, however. The disease is most serious

*Presented at the 37th annual meeting of the Virginia State Veterinary Medical Association, Winchester, Va., July 25-26, 1929.

in the first few months of life, the natural resistance increasing with age. In older birds the disease assumes the chronic form and birds may live some time before recovery or death.

The first indications you will notice will be that the young turk lags behind the rest of the flock. The wings are drooped and they present an unthrifty appearance. They are inclined to sit around instead of roving. They stop eating. The head is drawn in and oftentimes tucked under the feathers. The droppings are sulphur colored and in many cases there is a diarrhea, although this is not constant.

Diagnosis

When you see a flock of sick turkeys with the symptoms just mentioned, you may suspect blackhead, but there is only one way to be absolutely sure of the correct diagnosis. Perform a postmortem on a sick or dead bird. I do not know of a disease that is easier to diagnose than blackhead after you have become familiar with the lesions found in the liver and intestines of a bird that has died of the disease.

Lay the bird on its back and cut open the abdomen just in front of the vent. Extend the incision forward, cutting through the ribs on either side to the wings. The breast can then be doubled forward over the neck, exposing the heart, lungs, gizzard, liver and intestines in their natural positions.

The main changes in the organs in a case of blackhead are in the ceca, or blind pouches of the intestines, and the liver. The intestines of the turkey and chicken have a very peculiar arrangement. About 5 or 6 inches from the rectum there are two blind pouches or branches of the intestine. The blind ends of the pouches are toward the head. They are 4 or 5 inches long, about the size of a large lead pencil, and there is one on either side of the intestine. The disease originates in these blind pouches, one or both being affected. The parasite gains entrance to the inner lining and multiplies rapidly, penetrating deeply into the tissues, causing them to become greatly thickened. The pouches gradually become filled with a yellowish cheesy material about the consistency of cottage cheese. Frequently a core forms in the mass of material.

From the blind pouches the organisms are carried to the liver by the blood stream, where they set up severe pathological changes in that organ. The liver becomes greatly enlarged and dark red. Grayish or yellowish spots are noticed on the surface.

If the liver is cut open at these points it will be noted that the spots permeate the body of the organ. As a rule we find both the intestines and liver affected in the manner described, but occasionally only one organ will be affected. Characteristic lesions in either or both organs, however, are sufficient to make a diagnosis of blackhead.

Treatment

A great number of drugs and patent remedies have been recommended by investigators and laymen from time to time, for the prevention and cure of blackhead, but none have been proven of value. In diseases of large animals, where each animal is of considerable value, individual treatment can be attempted, but with poultry this is not practicable. In case of a large number in a flock it just can't be done. That is why we are continually advising prevention rather than cure in poultry diseases. Knowing the fact that it is necessary for the blackhead parasite to have the assistance of the chicken and the cecal worm in order for it to attack turkeys successfully simplifies the problem of prevention. Raise the turkeys and chickens away from each other.

Several of the parasitic diseases of both mammals and birds have two hosts. By destroying or eliminating one of the hosts we destroy or eliminate the disease-producing parasite. The protozoan parasite that causes malaria and yellow fever in man must spend part of its life in the body of one family of mosquitoes. We were able to build the Panamal Canal by eradicating malaria and yellow fever, but the mosquito had to be eradicated in order to get the two parasites.

The protozoan parasite that causes Texas Fever in cattle must spend part of its life in the body of a particular family of cattle ticks. The United States government and the various state governments are gradually pushing Texas fever into the Gulf of Mexico by destroying this particular family of cattle ticks. Other examples can be given. Just because the mosquitoes are eradicated to prevent malaria and yellow fever and cattle ticks to prevent Texas Fever does not necessarily mean that all the chickens must be destroyed to prevent blackhead in turkeys. A more economical as well as practical method has been worked out, *i.e.*, keep the turkeys and chickens separated and thus eliminate the cecal worm.

If the turkeys are raised with turkey hens, put them in a pasture or grain field away from the other poultry. Feed 2 per cent tobacco dust in the mash to keep the cecal worm under control. It would be a good idea to have fecal examinations made of the hens and if worm eggs are found, worm them or put them on tobacco dust treatment.

The safest method is to hatch the turkey eggs in an incubator and raise them in brooders in a clean field. Have the brooder made so it is portable and build a temporary fence on one side of the brooder. Every week or ten days move the fence to another side of the brooder. After the fence has been moved around the brooder, which takes four moves, change the location of both brooder and fence to another spot in the field.

This method has been published by *Capper's Farmer*, Topeka, Kansas, under the title of "Talking Turkey." I would advise all who are in a section where turkeys are raised to write for a copy. There need be no embarrassment for a veterinarian to write to an agricultural publication for this article, since the method was perfected and published by Dr. W. A. Billings, of the University of Minnesota.

To those of you with a superiority complex for surgery, you can prevent the disease by abligation of the ceca. The Missouri Experiment Station has been doing this successfully under experimental and farm conditions for two years with complete success. The method and results were published by A. J. Durant in the *North American Veterinarian.**

*x(1929), 2, pp. 52-55.

He Wore Gray Spats

When our little dog Daisy seemed poorly last week, we took her down to the Ellin Prince Speyer Hospital to get some professional advice on cod-liver oil. The visit was a disarming experience; it gave us a renewed sense of the benevolence of humanity as well as the dignity of animals. The reception room, white and sterile; the reception lady, starched and kindly; the other patients, soft-eyed and wet-nosed; the phone calls, "He has no temperature today!" were all part of a strong impression that this was the nicest place in New York. And when our turn came, and we ushered Daisy into the examination room and were greeted by a doctor who wore gray spats, it was too much happiness. Even Daisy was impressed, and took a turn for the better.

—*The Reader's Digest*

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

GASTRIC ULCER IN THE HORSE: REPORT OF A CASE*

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Ulcer of the stomach and duodenum is of rather frequent occurrence in man. One author reported that clinically ulcers may be noted in about 1 per cent of all general hospital admissions, and in about 4 per cent of all postmortem examinations.[2] Robertson and Hargis,[7] in an analysis of the examinations of 2,000 necropsies, found that ulcer or scars of ulcers were present in 18.9 per cent of cases; 7.05 per cent of these were gastric ulcers.

Gastric and duodenal ulcers have never assumed a significant part in veterinary medicine. Very little has been written on the subject. Law[4] mentioned gastric ulcer as being of infrequent occurrence in domesticated animals. The ulcers are most often seen in calves at the time they begin to eat coarse foodstuffs, such as hay and straw; trauma is an important factor. Hutyra and Marek[3] also stated that gastric ulcer is rare in domesticated animals; it occurs most commonly in the abomasum of calves. They further stated that gastric ulcer is rarely seen in adult cattle, horses, dogs and swine. Shillinger[9] recently reported an epizootic of spontaneous perforating gastric ulcers in ranch foxes.

In the lower animals, gastric ulcers generally occur near the pylorus. In the horse, erosions may occur in the cardiac portion of the stomach in connection with larvae of oestridae and *Spiroptera megostoma* which destroy and remove the cuticular covering, or with epithelioma or sarcoma growing in the gastric walls. Ulcers thought to be due to autodigestion are usually found in the right cul de sac. These generally are circular. Ulcers

*Received for publication November 11, 1929.

caused by infectious diseases are irregularly notched and marked by a mass of dark blood coagulated in their depth.

The hypotheses concerning the etiology of gastric ulcers in domesticated animals are many. Law stated that one of the simplest factors in the production of ulcers is the peptic juice. If the stomach becomes paralyzed while containing a quantity of secretion, it undergoes autodigestion. This affects particularly the lowest portion toward which the liquid gravitates, and the free edges and folds which are most exposed to its action. He further stated that irritant and caustic agents, by corroding or causing destructive inflammation of the exposed mucous membranes, may act similarly, especially in the monogastric animals. Trauma is considered the chief cause of gastric ulcer in calves. Such other causes as hot foods, gastric parasites, gastric catarrh, interrupted circulation (capillary thrombosis, arterial embolism or venous thrombosis), tumors, foreign bodies, tuberculosis, specific infections and various morbid nervous influences were mentioned. Hutyra and Marek stated that in animals peptic ulcers of the stomach always seem to result from acute ulceration or from hemorrhagic erosions. These erosions may be due to any of the causes mentioned.

Experimental investigations tend to show that mechanical injury is not the only factor in the cause of chronic ulcer. Acute erosions tend to heal with great rapidity. Morton,[6] in experiments on dogs, found that if he excised areas of mucosa alone and areas of mucosa and muscularis 2 cm. in diameter in the normal stomach the lesions healed rapidly. In two weeks, the entire denuded areas were completely covered with epithelium. The scars were small and often hard to find. If, however, the duodenal secretions were short-circuited during operation for the removal of the mucosal patches, ulcers developed in 50 per cent of cases. Mann and Williamson[5] produced peptic ulcers in dogs by surgically diverting the secretions which neutralize the gastric juice, as it leaves the stomach, to another portion of the intestine removed from the point of emergence of the acid.* Rosenow[8] has produced lesions in the stomach of normal rabbits by injecting cultures of organisms isolated from ulcers in man. Ulcers also occur in the stomach following extensive burns of the skin.

Hutyra and Marek stated that the symptoms of gastric ulcer in domesticated animals are generally those of chronic gastric

*Many other workers also have been successful in the production of experimental peptic ulcer.

catarrh or of atony of the first three stomachs. Horses may manifest symptoms of colic after the ingestion of food. Cattle may have attacks of gastric tympanites. In dogs, enlargement of the circumference of the abdomen, vomiting and icterus are manifested. Gradual emaciation, cachexia and complete ex-

FIG. 1. Raised scar on the peritoneal surface of the stomach.

haustion are common to all. Only occasionally does perforation occur.

The case which came under my observation occurred in a four-year-old gray gelding. I first saw the horse in September, 1928, at which time he had a severe attack of indigestion. He

was given symptomatic treatment, but failed to recover as promptly as was expected. He suffered from acute pain and discomfort for a week or ten days. At no time during this attack did the temperature go above 102°. Bowel movements were normal. After two weeks he had apparently recovered. However, he did

FIG. 2. Large elongated gastric ulcer near the pylorus, separated from the pylorus by isolated areas of fundic mucosa; small ulcer surrounded by this fundic mucosa.

not recover his weight completely even after a month. During the following eight months his appetite remained capricious and he had several recurrent attacks of colic. Relaxed bowel movements were common. At no time did he regain his original spirit and condition. The last month of his life he ate very little, be-

came emaciated and was unable to work. He was last observed alive, July 22, 1929. At this time he was suffering constant pain. Symptomatic treatment was instituted for the relief of pain. He died suddenly the following day.

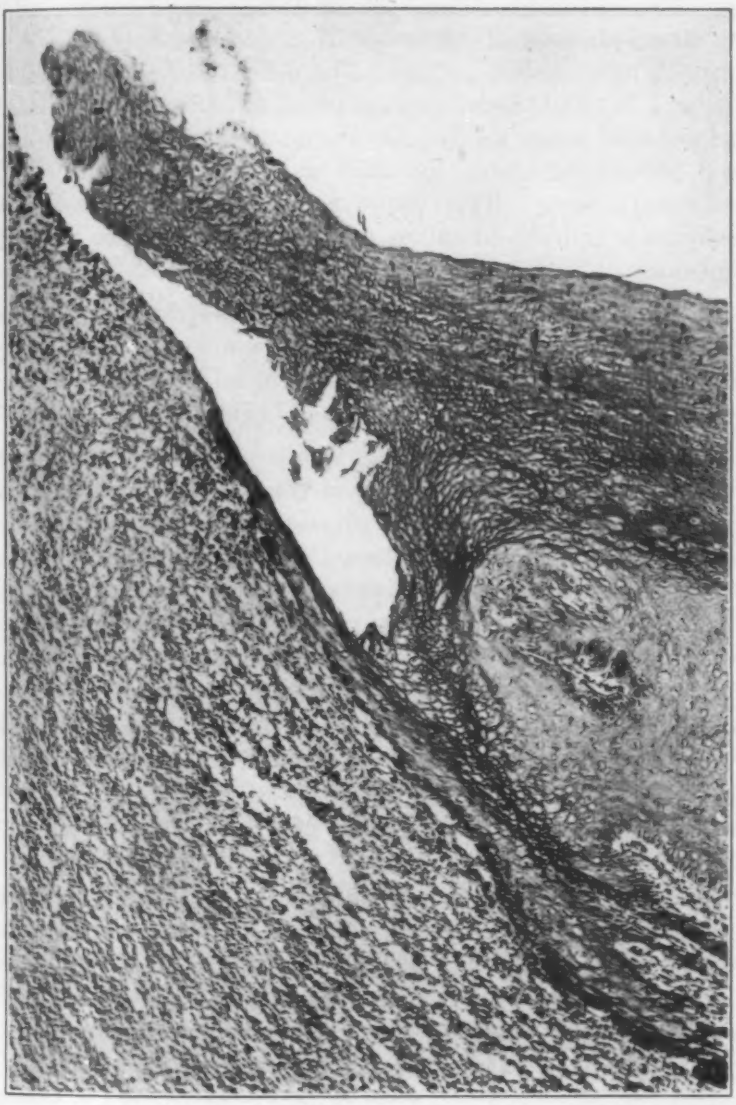

FIG. 3. Margin of large ulcer; healing changes and thin layer of epithelium growing out onto the granulation tissue bed. x 100.

Necropsy revealed slight traces of intestinal contents in the peritoneal cavity and general peritonitis. Careful search failed

to disclose any opening in the bowel or stomach. On careful inspection of the stomach a large raised scar was noted near the pylorus (fig. 1). The lesion was 36 by 4 cm. The stomach was opened and two large chronic ulcers were found (fig. 2). The largest was situated immediately on the cardia or fundus side of the margo plicatus. It measured 37 cm. in length and was 4 cm. in width in its widest portion. The other ulcer was irregular in cutline. It was 13.5 cm. in length by 3 cm. in width. This lesion was situated on the greater curvature and in the fundus; it had a deep perforating crater, by which gastric contents entered the peritoneal cavity. There were some adhesions between this perforation and the omentum. The largest ulcer also showed a scar along its full length on the peritoneal surface.

There were in the stomach about thirty oestridae larvae; some were lost in washing out the stomach. From gross examination it appeared that these may have been involved in the production of lesions. In several places there was beginning necrosis at the point of attachment of the larvae.

Microscopically, certain areas in these ulcers showed healing changes (fig. 3). This healing process was somewhat similar to that observed by Caylor[1] in man. There was a definite plateau of granulation tissue in the ulcer, and at the margin of the lesion was a thin layer of epithelium growing out on the granulation tissue. In the base of the ulcer were many vessels. A large number of these showed thickening of the walls and some contained canalized old thrombi. Hemorrhage and perivascular leukocytic infiltration were absent. The base of the defect was composed largely of connective tissue; it almost completely replaced the muscularis, only an occasional muscle bundle being demonstrable.

Because of the paucity of the literature of gastric ulcer in lower animals, one is led to believe that it is rare. However, it is possible that ulcers occur frequently, but because of the difficulty of diagnosis are not recognized in the living animal. Many of the so-called attacks of colic in horses may be the result primarily of such lesions. Another reason for the almost total disregard of gastric ulcer in veterinary medicine can be attributed to the fact that only a small percentage of domesticated animals dies of natural causes; most animals are slaughtered. Gastric lesions such as peptic ulcers are not looked for as a routine in post-mortem inspections in slaughtering establishments.

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PARATYPHOID DYSENTERY IN LAMBS AGAIN

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In 1923 we studied an outbreak of paratyphoid dysentery in feeder lambs, involving thirty thousand animals with a loss of 6.2 per cent.[1,2] Since that time we have repeatedly attempted to isolate the paratyphoid organism (*Salmonella aertrycke*) from lambs showing symptoms of dysentery, but with negative results. Now that we have again been successful it seems best to record the finding.

History: A lot of 1600 lambs was yarded at Barnhart, Texas, on October 10, and loaded on the following day. They were shipped to Amarillo, where they arrived at 7 a. m. on October 13, after having been on cars for 32 hours. They were unloaded and fed, but according to report they were put into cattle corrals where the hay was for the most part out of reach. They were reloaded at 3 p. m. and shipped to Denver, where they arrived the next day, after 30½ hours on the train. They remained in the yards at Denver all the next day and were then loaded out to Windsor, Colorado, arriving on October 16, six days after being yarded at Barnhart.

Symptoms and Lesions: On arrival at Windsor, five were dead in the cars and many were scouring. Two dead lambs were brought to our laboratory on October 23. Examination revealed carcasses in a moderate state of flesh, with wool around the anus and on the hind legs soiled with feces. The only significant lesions were a diffuse hemorrhagic colitis and cecitis, visible especially from the mucous surface.

Isolation of organism: Having just seen some cases of coccidial dysentery, a microscopic examination of the cecal content was made for coccidia, with negative results. Cultures were made on beef-infusion agar (pH 7.6) from the heart-blood and spleen of both sheep and from a lymph-gland of one of them. *Salmonella aertrycke* was obtained in pure culture from both heart-bloods, one spleen and from the lymph-gland.

The organism was motile, Gram-negative, produced acid from dextrose, mannitol, xylose, dulcitol, levulose, arabinose, sorbitol, rhamnose, maltose, galactose and inositol, and failed to do so from lactose, sucrose, salicin, dextrin and raffinose.

Loss: The place was visited on October 28, when it appeared that probably 5 per cent had been scouring and the owner stated that 29 had died. Only two animals showed symptoms of the disease on that date. One of these died on November 2, making the total loss 30 head.

Agglutination test: On November 3, blood was drawn from six animals that were believed to have recovered from the disease. This blood was run against the organism that we had isolated and also an antigen made from a strain that had been obtained from the 1923 outbreak, with the result as indicated in table I.

TABLE I—*Agglutination tests, using new and old strains of S. aertrycke*

No.	New Culture				Old Culture			
	1-25	1-50	1-100	1-200	1-25	1-50	1-100	1-200
1	—	—	—	—	—	—	—	—
2	+	+	+	+	+	+	+	±
3	+	+	+	—	+	+	—	—
4	+	±	—	—	+	—	—	—
5	±	—	—	—	+	±	—	—
6	+	+	±	—	+	±	—	—

SUMMARY

Another outbreak of paratyphoid dysentery in lambs is described. A railroad journey and difficulty in feeding appear to be predisposing causes. *Salmonella* aertrycke was isolated from two lambs. The blood serum of recovered lambs agglutinated the strain isolated, as well as the one from the previous outbreak.

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EPIDERMOPTIC SCABIES IN A HEN*

By W. A. JAMES, ROBERT GRAHAM *and* FRANK THORP, JR.

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University of Illinois, Urbana, Illinois

The first case of epidermoptic scabies in a hen observed by the writers was submitted for diagnosis by Dr. E. O. Smith, Princeville, Illinois. The affected fowl was an emaciated adult Rhode Island Red hen. In the farm flock from which this fowl originated

FIG. 1. Gross lesions of epidermoptic scabies in fowls.

there were between 300 and 400 chickens, yet only one fowl in the flock was visibly affected. No prophylactic treatment was administered to the remainder of the flock, and no new cases were

*The parasite was identified by Doctor Albert Hassall, of the Zoological Division, Bureau of Animal Industry, Washington, D. C., as belonging to the species Epidermoptes.

observed for a period of six months in this or adjoining flocks of the locality.

Upon external examination, hard, brownish yellow, elevated, circumscribed brown scabs, varying in size from a pea to a small hazelnut, were found on the skin and legs (fig. 1). Some of the scabs were removed and scrapings made of the underlying cutaneous tissue. Microscopic examination of smears revealed a mite resembling psoroptes or sarcoptes (fig. 2). The air-sac and subcutaneous-tissue mites were first suspected, along with the possibility of a mite infestation from heterologous host. At

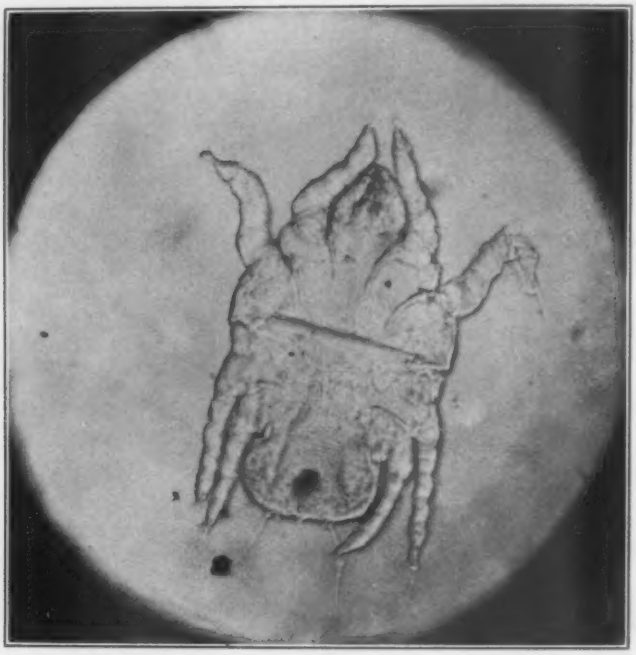

FIG. 2. The epidermoptic mite from lesions shown in fig 1.

autopsy the air-sacs and subcutaneous tissues were free from parasites. No other gross pathologic conditions except emaciation and parasitic dermatitis were found.

The scabs consisted of dried exudate, which, under the microscope, appeared as a homogeneous, structureless material taking the eosin stain faintly. Sections of the skin and subcutaneous tissue in the parasitized area upon microscopic examination showed a definite hyperplasia with areas of acute infiltration. In the skin hyperplasia tissue beneath the scab, areas

of fibrin were present. No mites were found in microscopic sections of the skin.

TREATMENT

Little is known regarding the treatment of this type of scabies in fowls. Avian epidermoptic scabies is a rare condition in the Corn Belt as judged by the dearth of clinical material and the absence of reports of the disease in veterinary literature.[1] Since the history of the disease in this flock suggests that it is but mildly contagious, isolated cases in fowls should be destroyed and the premises disinfected. If several fowls in an infested flock suffer from the disease, the usual lime-sulphur or nicotin dips might be given a trial, after the scabs are removed.

REFERENCE

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CAPILLARIA ANNULATA IN QUAIL*

By E. F. THOMAS, *Assistant Veterinarian*

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September 16, 1929, a quail was brought to the laboratory for a postmortem examination. The owner stated that several birds had died in the small flock which he has kept in captivity. The history revealed that the prevailing symptom was a diarrhea, somewhat whitish in color. The birds died quite soon after the symptom of diarrhea had begun.

A heavy accumulation of mucus was found in the crop and esophagus. Numerous lemon-shaped ova were found in this mucus. Underneath the mucus and extending into the mucosa were numerous slender, thread-like, colorless worms, which were very difficult to remove. The worms were classified as *Capillaria annulata*. This parasite was reported by Cram,[1] in 1926, for the first time in this country. Since that time it has proven to be fairly common in many of our domestic birds.[2]

No satisfactory treatment is known. The life history of this parasite is also unknown. In addition to the infestation of *Capillaria annulata*, the quail was also heavily infested with the tapeworm, *Davinea cesticillus*. It is believed that the infestation with the latter parasite caused the death of the bird.

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*Received for publication, December 2, 1929.

A PRACTICAL AND CONVENIENT METHOD OF HANDLING BLOOD SAMPLES IN THE FIELD*

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Only the laboratory technician can appreciate to the fullest extent the importance of properly drawn and handled blood samples in obtaining clear serum for various serological tests. A

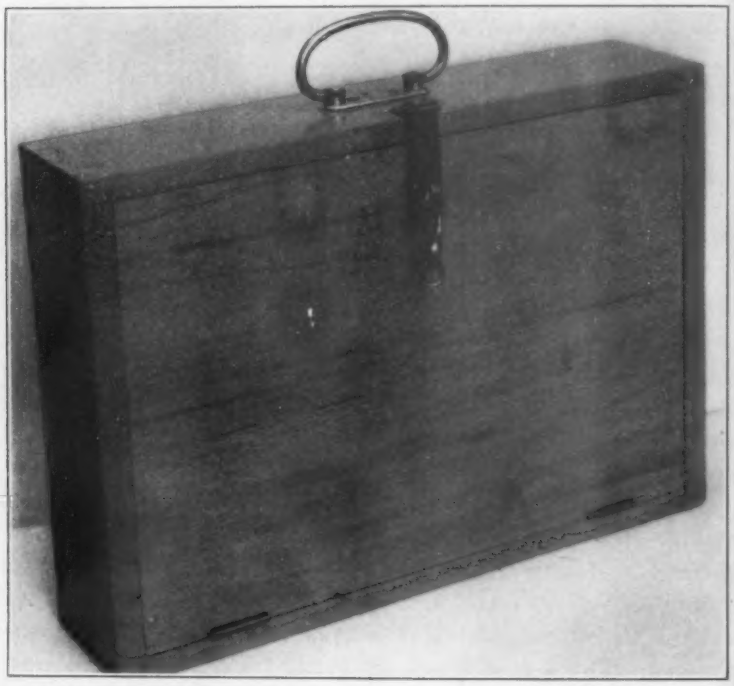

FIG. 1. Box for carrying blood samples (closed).

sample drawn in a proper container and properly handled can be in the best of condition to test within 15 to 20 hours following the drawing of the sample. On the other hand, as any person who has had experience in testing samples of blood from a wide variety of sources will agree, blood samples can be drawn into such containers or handled in such manner as to make it quite impossible to secure clear serum from the sample without breaking up the blood clot in each container and letting it stand for 24 to 48 hours

previous to testing, or by centrifuging. In the laboratory where hundreds of samples are tested weekly, this procedure is a great inconvenience, in fact, most impractical.

We have found that a 10-cc, non-lipped homeopathic vial is most satisfactory to handle, both in the field and in the laboratory. These vials are 1.5 cm. in diameter and 5.5 to 6 cm. in length. The straight vial is more easily cleaned. Although 4 cc of blood will yield sufficient serum (2 cc), it is necessary that a vial as above described be used. In order to obtain the quickest

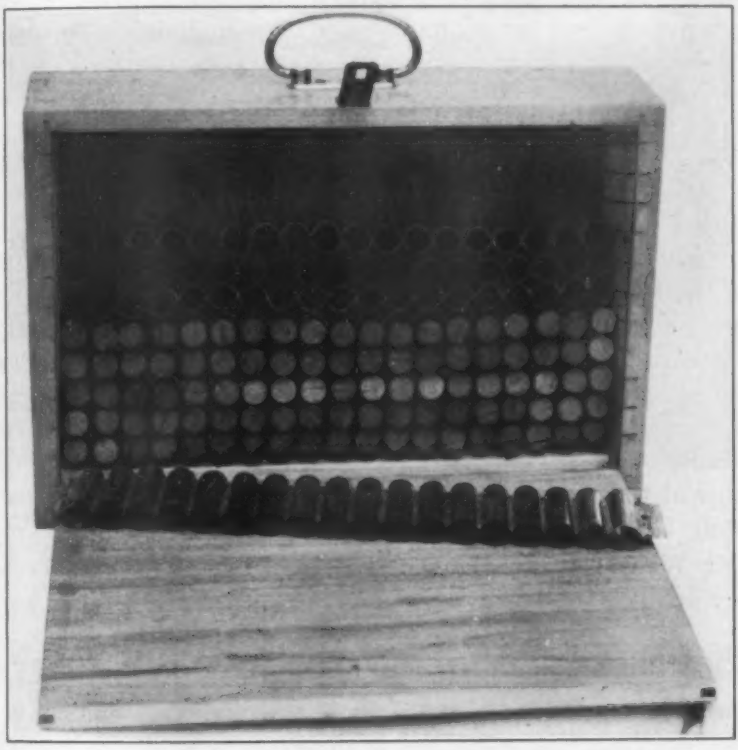

FIG. 2. Box open, showing arrangement of vials.

and greatest production of serum in the most convenient condition for pipetting, the vial, containing approximately 4 cc of blood, should be placed on an incline such as will allow the blood to run nearly to the stopper. As soon as the sample has been drawn, it should be placed in the correct position and allowed to remain undisturbed until the clot is sufficiently firm to avoid breaking.

*Journal article No. 23, N. S.

If labels are used on the vials, they should not extend more than half-way around the vials and in slanting the vial, after taking the sample, the label should be on the under side. The purpose of this is to enable the technician to watch the point of the pipette as he lowers it into the serum.

To devise a convenient and practical method of properly handling blood samples, under all sorts of barn conditions, may be somewhat of a problem, especially to the practitioner who does a great deal of bleeding. We have been confronted with that problem for some time. During this time various methods and devises were used but all had one or more undesirable features. In a determined effort to overcome these disadvantages, a box was constructed which has proved satisfactory beyond expectations.

The photographs will suffice to give a general idea of the construction, which is all that is necessary since each one will desire a box of such capacity as will best suit his requirements.

In figure 1 is shown the closed box, measuring 3½ x 10½ x 18 inches, with a capacity of 210 vials.

Figure 2 shows the open box, giving a view of five lower racks filled with vials, three empty racks in place and three removed. In the foreground is shown a single rack, so as to demonstrate the details more clearly. Each rack is composed of two strips of specially corrugated, light tin, 1¾ inches in width, soldered together at the ends. The racks are set in at an angle of approximately 17 degrees.

Mr. Wm. Klever, of the Department of Bacteriology, deserves much credit for assisting in the construction of the blood-vial box.

INCOMPLETE SEXUAL DEVELOPMENT IN A HEIFER

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This case of incomplete sexual development occurred in a grade Shorthorn heifer. At one year of age, the first and only estrual period occurred, at which time she was served by the herd bull. Evidence of pregnancy did not develop and the animal was slaughtered following a rectal examination which revealed a rudimentary reproductive system. Autopsy findings were as follows: The cervix and body of the uterus were represented by

a slender band of connective tissue, two inches in length; the horns of the uterus consisted of two closed tubes, one and one-half inches in length by one-half inch in diameter. These tubes were situated in the form of a V, and were distended with fluid. About two inches anterior to the horns, imbedded in a rudimentary broad ligament, were two yellowish structures which probably represented ovaries. As it was decided to keep the specimen intact, no microscopic studies were made.

Five months prior to the appearance of estrum and for a period of four months thereafter, the blood of this animal gave negative

FIG. 1. The uterus of a heifer twenty-seven months of age.

results with the agglutination test for infectious abortion. On the latter date, a large dose of *Bact. abortus* organisms was administered per os. Thirty days later, a doubtful reaction was obtained with the agglutination test. Subsequent tests were negative, and at two years of age a second dose of *Bact. abortus* was administered per os. Sixty and ninety days later, strong positive reactions were obtained with the agglutination test. Evidently, susceptibility to infection with *Bact. abortus* was not dependent on a complete development of the reproductive organs.

Southeastern States Veterinary Medical Association

The Louisiana Veterinary Medical Association and the Mississippi State Veterinary Medical Association will meet in conjunction with the Southeastern States Veterinary Medical Association, in New Orleans, February 10-11, 1930. The Gulfport meeting of the latter organization has been called off.

ABSTRACTS

EXPERIMENTAL RICKETS. T. Skaas and K. Haeupl. Virchow's Arch. f. Path. Anat. cclxxi (1929), p. 100. Abst. in Arch. Path., viii (1929), 3, p. 506.

Young dogs were fed on Mellanby's rickets diet. After three weeks, the first signs of rickets appeared. Addition of 40 cc of cod-liver oil daily effected a rapid cure. The bone thickenings disappeared, but the curvatures persisted. The phosphorus and calcium metabolism of the rachitic dogs was negative. The phosphorus and calcium content was low. After giving cod-liver oil, the serum phosphorus rose more quickly than the serum calcium. The bone of the cured animal contained less phosphorus and calcium than that of the control animal (which had 10 cc of cod-liver oil daily). In some dogs the phosphorus metabolism was more damaged; in others, the calcium metabolism. There seem to be different forms of rickets. Large doses of phosphorus and calcium led to an increase of calcium and phosphorus in the serum; but the bones of these dogs did not contain more phosphorus or calcium than those of the controls. Excessive formation of lime-free bone in the whole skeleton is characteristic of rickets. The authors conclude that true rickets can be produced by a deficient diet and cured by cod-liver oil.

DIFFERENTIATION OF HEMOLYTIC STREPTOCOCCI OF HUMAN AND OF DAIRY ORIGIN BY METHYLENE BLUE TOLERANCE AND FINAL ACIDITY. Roy C. Avery. Jour. Exp. Med., 1 (1929), p. 463.

Three groups were distinguished among 138 strains of hemolytic streptococci. The differences in the groups were based on their dye sensitiveness and on the final hydrogen-ion concentration of cultures. The groups are: 1. Human parasitic strain, defined by a final pH range of 5.2 to 5.0 and by failure to reduce methylene blue (1 to 5000) in milk. 2. Bovine strains, parasitic in the udder, characterized by a final pH range of 4.5 to 4.2 and by failure to reduce methylene blue (1 to 5000) in milk. 3. Saprophytic strains, characterized by a final pH range of 4.5 to 4.2 and by ability to reduce methylene blue.

A Paralytic Disease of Guinea Pigs Due to the Tubercle Bacillus. Richard E. Shope and Paul A. Lewis. Jour. Exp. Med., 1 (1929), 3, p. 365.

The experimental data collected during this study of a transmissible type of paralysis developing in tuberculous guinea pigs indicate the condition to be a true tuberculous meningitis. Typical tubercle bacilli were readily demonstrable in sections of the meninges from animals with the disease, and cultures of brain tissue on Dorset's egg medium usually yielded a growth of the organism. In view of the much discussed question of the filtrability of the tubercle bacillus, the observations of the authors concerning the failure of the organism to pass a Berkefeld N filter are of interest. No animal inoculated intracerebrally with brain emulsion failed to develop meningitis, while no animal injected with a Berkefeld filtrate of brain emulsion has developed tuberculous meningitis or any other form of tuberculosis.

Transmission of Fowl-Pox by Mosquitoes. I. J. Kligler, R. S. Muckanfuss and T. M. Rivers. Jour. Exp. Med., xlix (1929), p. 649.

Culex and Aedes mosquitoes are capable of transmitting fowl-pox from diseased to healthy susceptible chickens. The mosquitoes remain infectious for at least fourteen days following a meal on diseased fowls.

A Fatal Infection of Chicks Due to Bacilli of the Paratyphoid Group. Phillip R. Edwards. Jour. Inf. Dis., xlv (1929), 3, p. 191.

The author describes an epizootic occurring in a flock of 2000 baby chicks. The outbreak became apparent when the chicks were but three days of age and continued until a mortality of 25 per cent was reached. The symptoms closely resembled those of bacillary white diarrhea, an extremely edematous condition of the abdominal organs being most pronounced. Two organisms of the paratyphoid B group (*B. aertrycke* and *B. anatum*) were isolated from the chicks autopsied. The source of the infection was undetermined.

Maternal Control of the Placental Glycogen. A. St. G. Huggett. Jour. Physiol., lxvii (1929), 4, p. 360.

The author, working with rabbits, found that as far as the maternal stimuli are concerned, the placental glycogen is not very

susceptible to changes and is not influenced by ordinary factors of maternal metabolism. However, on gross demands on the carbohydrate stores over a long period of time, the glycogen slowly disappears, though never entirely under the conditions of the experiment. The glycogen of the placenta is much more stable than that of the liver. Factors which lower or raise the blood pressure have no effect upon it. Ether, amytol, and repeated doses of insulin cause a slight fall. The glycogen of the maternal portion of the placenta appears to be a reserve for the fetus which the mother normally can not draw upon except in extreme cases of disordered metabolism over a prolonged period.

THE SKIN AS A PORTAL OF ENTRY IN BRUCELLA MELITENSIS INFECTIONS. Albert V. Hardy, Margaret G. Hudson and Carl F. Jordan. Jour. Inf. Dis., xlv (1929), 4, p. 271.

Observing that undulant fever occurred more frequently in packing-house employes and those in more intimate contact with animals on farms, the authors investigated, experimentally and epidemiologically, the skin as a portal of entry of *Br. melitensis* infections. Two strains of human origin were used. Groups of guinea pigs in which the abdomen was shaved with abrasions, shaved without abrasions, and with the hair clipped, were exposed by applying the organisms to the area. The percentage of infections as determined by the agglutination test and isolation of the organism was 100 per cent, 90 per cent, and 75 per cent, respectively. A fourth group fed per os resulted in 22 per cent infection, it being found, however, that the proportion infected by feeding varies with the dose. An epidemiological study of the occurrence of the infection in packing-house employes and persons living on farms, with the presence of agglutinins as an indication of infection, with or without clinical manifestations, justifies the opinion that the skin of humans is a ready portal of entrance of *Brucella melitensis*. It is pointed out that this avenue of infection may be common among laboratory workers. The authors conclude that the normal skin of guinea pigs is more vulnerable as a portal of entry than is the digestive tract and that the epidemiological evidence indicates that the same is true of humans. Since the organisms gain entrance without causing any local lesions, the probable portal can be determined only by considering carefully the types of exposure, the dosage, and the resistance to invasion at the different portals. The authors believe that in-

gestion is not a satisfactory explanation for the natural and ready transmission of contagious abortion among animals and that more consideration should be given to the skin as a portal of entry.

THE PATHOGENICITY OF THE SPECIES OF THE GENUS BRUCELLA FOR MONKEYS. I. Forrest Huddleson and E. T. Hallman. Jour. Inf. Dis., xlv (1929), 4, p. 293.

Infective agents consisted of infective cows' milk, infective stomach exudate of an aborted fetus, and pure cultures of the three species of the genus Brucella of animals as well as human origin. Nineteen monkeys of the species *Macacus rhesus* were used. Agglutinins in the blood serum were considered presumptive evidence of infection. Gross and microscopic lesions appearing in the lungs, liver, kidneys, spleen, lymph-glands, gastrointestinal tract and reproductive organs are recorded. The experiments indicate that *Brucella abortus* (Bang) does not possess a high degree of virulence for monkeys. Only after repeated feedings of infective milk was infection produced in one of three monkeys. Strains from humans produced slight infection in two monkeys, no infection in the third. *Br. suis*, regardless of source or origin of the strain, possessed a high degree of virulence for monkeys, one exposure being sufficient to infect each of the seven monkeys used. *Br. melitensis* proved less virulent than *Br. suis*. The temperature curves could not be relied upon for diagnosis. The difference in the degree of virulence of the species of Brucella and the difference in the degree of susceptibility of the monkeys suggest an explanation for the difference in the susceptibility of human beings towards these organisms.

A FILTRABLE FIBRO SARCOMA OF THE FOWL. A. M. Begg. Jour. Exp. Path., viii (1929), 5, p. 322.

A hard, slow-growing fibro sarcoma of the fowl has increased its rate of growth during transplantation and became softer. The histological appearances have changed from those of a hard fibroma to be a more cellular and sarcomatous type. It can now be propagated by cell-free filtrates and by dried material, but it has not been proven that it was not so originally.

THE GROWTH OF PASTEURELLA AVICIDA IN THE BODY OF THE FOWL. L. D. Bushnell and V. D. Foltz. Jour. Inf. Dis., xlv (1929), 4, p. 308.

Pasteurella avicida appears to have an organ virulence for blood and is able to grow irrespective of the manner in which introduction is made. The organisms possess so little toxicity that they do not call forth any marked response on the part of the host. The exudation into the lungs may be due to serotoxins or some product due to the interaction of the bacteria and serum or tissue cells which does not appear in artificial culture. The exact cause of death in fowl cholera, as indicated by the clinical symptoms, seems to be oxygen starvation.

The Simultaneous Production of Two Hormones by the Corpus Luteum. Robert T. Frank, R. G. Gustavson, Helen McQueen and Morris A. Goldberger. Amer. Jour. Physiol., xc (1929), 3, p. 727.

The authors have demonstrated that the corpus luteum is able to and does elaborate two distinct and different hormones simultaneously, both of which are necessary for the complete function of the genital tract. One, the ether fraction, is designated as the female sex hormone, which is secreted by the growing and mature follicle, the corpus luteum, as well as by the placenta (the three forming the gestational gland). It is the anabolic hormone. The other, the aqueous corpus luteum hormone, is the cycle-inhibiting and "nidatory" hormone.

Tuberculosis of the Common Crow. Chas. A. Mitchell and R. C. Duthie. Amer. Rev. Tuber., xix (1929), p. 134. Abst. in Arch. Path., viii (1929), 5, p. 849.

The incidence of tuberculosis in wild animals and birds in their natural state is largely a matter of conjecture and is governed in all probability by their relative chances of exposure rather than by any marked lack of susceptibility. In the case of Corvidae and birds of similar habits, contact with man and domesticated animals can not be excluded. These birds might become infected in a number of ways through contact with tuberculous poultry, domesticated animals and their dejecta, infected waste products from garbage dumps and country slaughter-houses, and unburied carcasses of diseased animals. The susceptibility of Corvidae to mammalian tuberculosis is at present unknown. The ability of the crow to transmit infection to other birds or animals remains to be shown.

STUDIES OF THE BLOOD CHANGES OCCURRING IN YOUNG AND OLD DOGS DURING CUTANEOUS AND ORAL INFECTION WITH THE DOG HOOKWORM, ANCYLOSTOMA CANINUM. Merritt P. Sarles. Amer. Jour. Hyg., x (1929), 3, p. 693.

Both oral and cutaneous infection of adult dogs caused a marked increase in the total number of leukocytes in the blood. There was an immediate decrease in the number of eosinophiles following both oral and cutaneous infection, succeeded by a gradual increase which produced an eosinophilia of 17 to 42 per cent on the tenth to fourteenth day after infection. This change in the number of eosinophiles in the blood was very constant and similar in the six adult dogs studied. In young dogs cutaneous and oral infection failed to produce a marked eosinophilia and a leukocytosis was not a constant occurrence. Following cutaneous and oral infections of approximately 20,000 larvae, less than 25 worms developed in the intestines of adult dogs, and no significant decrease resulted in the grams of hemoglobin or number of red blood corpuscles. Cutaneous and oral infections of approximately 10,000 larvae produced, in young dogs, infestations of over 1000 worms and caused an acute and fatal anemia in the second week after infection.

THE REACTION AND SUSCEPTIBILITY OF DOGS OF DIFFERENT AGES TO CUTANEOUS INFECTION WITH THE DOG HOOKWORM, ANCYLOSTOMA CANINUM. Merritt P. Sarles. Amer. Jour. Hyg., x (1929), 3, p. 683.

In young dogs the penetration of the larvae either produced no local reaction or a very slight and transient one. The larvae migrated rapidly from the skin, through the lungs to the intestine and only a small number were found in the skin later than the first day after infection. The percentage development of the larvae following cutaneous infection of young dogs was much less than for oral infection of dogs of the same age. In old dogs there was an immediate local reaction to the penetration of the larvae, followed by a violent and prolonged inflammation. Sections of the skin showed evidence of the destruction of the hookworm larvae during this process. The larvae were retained in large numbers in the skin, although some succeeded in undergoing a delayed migration to the intestine. In old dogs the percentage development was very small by both methods, showing the

presence of age resistance. The persistence of undeveloped larvae in the intestines of dogs followed cutaneous infection.

THE EFFECT OF AGE AND SIZE OF INFESTATION ON THE EGG PRODUCTION OF THE DOG HOOKWORM, ANCYLOSTOMA CANINUM. Merritt P. Sarles. Amer. Jour. Hyg., x (1929), 3, p. 658.

The egg-production of the worms was found to be much less in large infestations than in small ones. This was found to be true for both experimental and natural infections. The difference could not be explained by differences in the size of the worms. The egg-production of the worms increased definitely only during the first month of an infestation and increased more in small infestations than in large ones.

THE LENGTH OF LIFE AND RATE OF LOSS OF THE DOG HOOKWORM, ANCYLOSTOMA CANINUM. Merritt P. Sarles. Amer. Jour. Hyg., x (1929), 3, p. 667.

The natural duration of infestations of *A. caninum* in young dogs varies considerably from one dog to the next. Most of the hookworms are lost in the first half-year, although a few may persist for nearly two years. The hookworms are lost at a constant rate. Dogs which have harbored hookworms for a long period of time are much more resistant to infection than young dogs. Most of this resistance is certainly due to age. The experiments suggest that a minor part of the resistance may be acquired.

RELATION OF ESTRUS HORMONE TO NYMPHOMANIA OR PERSISTENT ESTRUS OF COWS. W. Frei and E. Lutz. Orig. Virchow's Arch. f. Path. Anat., cclxxi (1929), p. 572. Abst. in Arch. Path., viii (1929), 5, p. 838.

Of the various substances that have been described as having an estrus-producing action, Frei and Lutz consider that only the follicle fluid or the cells of the follicle wall contain the true hormone. They doubt that true and complete estrus has been caused by some of the substances used. The occurrence of the hormone in the corpus luteum and placenta, as described by many, is paradoxic. The authors believe the corpus luteum absorbs, stores and renders inactive the hormone formed by the follicle. Nymphomania of cows is a condition of persistent sexual

desire or estrus. Manual rupture through the vagina of an enlarged ovarian follicle sometimes overcomes the condition. The vaginal smear of nymphomaniac cows contains epithelial cells and leukocytes, but no hornified epithelium. The authors conclude that nymphomania is due to the persistence of unruptured follicles in the ovary. Since no corpora lutea are formed, the follicle hormone acts continuously. Nymphomania may go into anaphrodisia when the ovary becomes completely cystic or atrophic.

REVIEW

KENNEL BUILDING AND PLANS. Will Judy. Second edition. 56 pages. Illustrated. Judy Publishing Company, Chicago, 1929. $1.00.

The first edition of this handy little book appeared in 1927 and was reviewed in the JOURNAL for January, 1928. The second edition is just twice the size of the first, and the publishers claim that it is the only book of its kind. It appears to be complete, practical and sufficiently detailed to be really useful. No expense has been spared in the illustrations.

Veterinary Institute with Clinic and Polyclinic, Court View, Windmill Way, Leipsic, Germany.

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Inheritance of Chalk-Face in Merino Sheep. B. L. Warwick and D. S. Bell. Reprint from Rec. Proc. Ann. Meet. (1928) Amer. Soc. Anim. Prod.

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Genetic Tests in Animal Breeding. B. L. Warwick. Reprint from *Bimonthly Bul. 138, Ohio Agr. Exp. Sta.*, Wooster, Ohio (1929), pp. 94-96.

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Acidosis in Heat Stroke. A Report of Two Cases in Horses Successfully Treated. George P. McNeill, Jr., U. S. A. Reprint from *Military Surgeon*, Sept., 1929, pp. 371-375.

The Relation Between Fodder and Quantity of Milk. Takeda Uyeki. (S. Hata, Tokyo, 1929.) pp. 90.

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The Treatment of Undulant Fever with Vaccine. Report of Ten Cases. Fred E. Angle. Reprint from *Jour. Kans. Med. Soc.*, October, 1929.

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Improved Reindeer Handling. (Cir. 82. U. S. Dept. Agr., Washington, D. C.) pp. 18.

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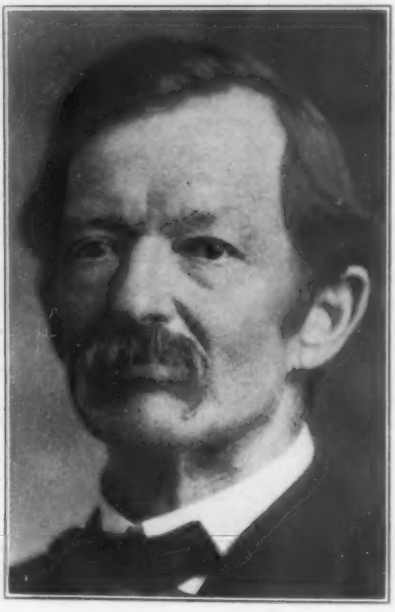

DR. C. A. CARY

Member of the Executive Board of the A. V. M. A., representing District No. 4.

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

Captain F. H. K. Reynolds has been assigned to duty at Fort Sam Houston, Texas, upon completion of his present tour of foreign service with the Medical Research Board in the Philippine Department.

Captain Robert P. Kunnecke, who has been under observation and treatment at Walter Reed Hospital for some time, has been ordered before an Army retiring board.

Veterinary Reserve Corps

New Acceptances

Allen, James Sidney	Captain	420 Maple St., Idaho Falls, Idaho
Bailey, Herbert Glenn	Captain	2116 Bull St., Savannah, Ga.
Bigelow, Fred Carlton	Captain	34 W. Pomfret St., Carlisle, Pa.

Promotions

Lynch, David Joseph	to Lt. Col.	Shepherd, Mich.
Muldoon, Wm. Edward	" Lt. Col.	32 W. 7th St., Peru, Ind.
Robertson, Robert James	" Major	7756 S. Marshfield Ave., Chicago, Ill.
Seute, Wm. Herman	" Capt.	9th & Main Sts., Pleasanton, Kans.
Waldron, Adolphus Wm.	" Capt.	815 N. Washington Ave., Cookeville, Tenn.

Thirteenth Officers' Basic Course

The Surgeon General of the Army has stated that he would like to have civilian veterinarians look upon the Veterinary Corps of the Regular Army as an instrumentality for the upbuilding and advancement of the veterinary profession, subject, of course, to the limitations imposed by military customs and jurisdiction.

One of the functions of the Army Veterinary School is that of investigation and research, with a view to the better control of diseases affecting animals and improvement of the present methods of animal management. The veterinary officers are anxious to gain and maintain contact with such activities outside of the Army and to make use of their facilities to further the cause.

Among the classes of officers from which students will be selected and detailed for courses at the Army Veterinary School are officers of the Reserve Corps of the National Guard and of the Veterinary Corps Reserves, eligible in accordance with instructions of the War Department. National Guard and reserve officers of other departments of the Army attend the Army Service School of the branch to which they are assigned. It is hoped that in the future, veterinary officers of that class may be able to attend the Army Veterinary School.

The accompanying photograph shows those who took the thirteenth Officers' Basic Course at the Army Veterinary School, Army Medical Center, in Washington, recently:

Front row (left to right): 2nd Lt. Harvie R. Ellis; Capt. Frank M. Lee; 1st Lt. Gustavo Rodriguez y Xiques, of the Veterinary Corps, Cuban Army; Capt. Gardiner B. Jones; Capt. Charles M. Cowherd.

Middle row: 2nd Lt. Arvo T. Thompson; Capt. Gerald W. Fitz Gerald; Capt. George W. Brower; Capt. Harry J. Juzek, Executive Officer and Instructor in Department of Meat and Dairy Hygiene.

Rear row. Maj. Geo. H. Koon, Commandant, The Army Veterinary School and Instructor in Department of Meat and

VETERINARY OFFICERS WHO TOOK POSTGRADUATE WORK

Dairy Hygiene, Department of Forage Inspection and Department of Clinical Medicine, Surgery and Animal Management; Maj. Raymond A. Kelser, Officer in Charge of the Veterinary Laboratory Section of the Army Medical School and Instructor in the Department of Preventive Medicine and Clinical Pathology, Department of Roentgenology, of the Army Medical School; Capt. Arthur D. Martin.

Lts. Thompson and Ellis are recent appointees to the Veterinary Corps of the Regular Army. Students attend the Army Veterinary School for five months and those who successfully complete the course are certified as accredited for tuberculin testing by the Bureau of Animal Industry and are licensed as

Federal Hay Inspectors by the Bureau of Markets, U. S. Department of Agriculture.

The veterinary officers of the Army are trying to make this school the very best postgraduate course in veterinary science that can be secured, with especial adaptation to the military needs. The ranking member of the above class has completed nearly fourteen years of service and the lowest in rank has not yet completed six months service. The course primarily is for newly appointed officers, but the schedule, as now drawn, covers subjects that can be profitably pursued by any veterinary officer.

International Veterinary Congress

A great deal of interest is being shown in the European tour in connection with the International Veterinary Congress to be held in London, this year. Among those who have already made reservations for the tour are: Dr. and Mrs. John R. Mohler and daughter, Washington, D. C.; Dr. and Mrs. A. T. Kinsley and son, Kansas City, Mo.; Dr. and Mrs. H. E. Curry, Kansas City, Mo.; Dr. and Mrs. A. F. Schalk, Fargo, N. Dak.; Dr. and Mrs. J. F. Devine and daughter, Goshen, N. Y.; Dr. and Mrs. E. B. Ackerman, Huntington, Long Island, N. Y.; Dr. and Mrs. Adolph Eichhorn and son, Pearl River, N. Y.; Dr. and Mrs. J. O. F. Price, Algona, Iowa; Dr. F. A. Grenfell, Pearl River, N. Y.

Veterinary Institute with Clinic and Polyclinic, Main Front, Linne Street, Leipsic, Germany.

MISCELLANEOUS

"Sleepy Grass" Causes Horses to Doze

"Sleepy grass," a stock-poisoning plant so named because of its effect on horses, grows in many locations in the semi-arid and arid parts of the Southwest, but as far as has been learned, it is poisonous in its effect only in New Mexico and in only two counties there. Experimental work done with the grass is reported upon and discussed in Technical Bulletin No. 114-T of the U. S. Department of Agriculture, "Sleepy Grass (*Stipa Vaseyi*) as a Stock-Poisoning Plant," by Dr. C. Dwight Marsh and A. B. Clawson, specialists on stock-poisoning plants. The bulletin is technical in nature and intended primarily for veterinarians and scientific workers.

Sleepy grass is known to science as *Stipa vaseyi*. Other species of Stipa are known to be poisonous in South America, China, and elsewhere. Sleepy grass is a stout, upright, perennial grass, with narrow, mostly flat, leaves and narrow green or tawny flowerheads. It has been reported as poisonous to horses, cattle and sheep, but in the feeding experiments conducted by the Department of Agriculture only horses were affected seriously, cattle were not much affected, and sheep, although slightly affected, did not show drowsiness.

In the experiments, less than 1 per cent of the weight of a horse in green grass, or the equivalent in hay, caused drowsiness, fever and low pulse. Affected animals often lay with their necks and heads stretched out on the ground or with their chins touching the ground. The animals were affected in their gait, some dragging their hind feet when they could be induced to move. The grass produced profound slumber but not death, and recovery apparently was complete after about 24 to 36 hours. The grass grows widely in the Southwest, but definite cases of sleepy-grass poisoning are reported from only two general localities, in Otero and Lincoln counties, New Mexico.

The bulletin may be obtained free, from the office of Information, Department of Agriculture, Washington, D. C., as long as there is a supply available for free distribution.

Ohio State Student Chapter Gets Publicity

The accompanying photograph shows a float which was made by the members of the Ohio State University Student Chapter

of the American Veterinary Medical Association and entered in a parade that preceded the football game between the teams representing the University of Illinois and Ohio State University this past season. The float was one of thirty-six entered in the parade, which was held on Homecoming Day. On this occasion there is always keen competition among the fraternities and sororities at Ohio State University to see which house can be decorated the most artistically. Recently an opportunity was

FLOAT OF O. S. U. STUDENT CHAPTER OF THE A. V. M. A.

afforded all non-Greek letter organizations on the campus to see what they could do in a parade. Although the float entered by the O. S. U. Student Chapter did not win first place, it was of such merit that the photograph was reproduced on the front page of the *Columbus Dispatch* the evening of November 23. Carl States, a junior student, made the emblem and the blue cross shown on both sides of the float. The members of the O. S. U. Student Chapter are to be commended for their progressiveness on such an occasion.

Order a Binder

Take advantage of the opportunity, when remitting your 1930 A. V. M. A. dues, to order a binder, or a set of them, for the JOURNAL. See advertising section for prices and details.

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1929-1930

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	Term Expires
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E. A. Benbrook, Iowa State College, Ames, Iowa	1930
N. S. Mayo, c/o Abbott Labs., North Chicago, Ill.	1932
Reuben Hilty, 624 Huron St., Toledo, Ohio	1933
C. D. McGilvray, Guelph, Ontario, Canada.	1934

Committee on Legislation

	Term Expires
J. P. Turner, *Chairman*, 1357 Kennedy St. N. W., Washington, D. C.	1930
J. L. Axby, Lawrenceburg, Ind.	1931
F. E. Murray, 326 Federal Bldg., Salt Lake City, Utah	1932
Cassius Way, 452 Lexington Ave., New York, N. Y.	1933
C. A. Cary, Auburn, Ala.	1934

Committee on Program (Ex Officio)

H. Preston Hoskins, *Chairman*, 716 Book Bldg., Detroit, Mich.
H. E. Kingman, Colo. Agr. College, Fort Collins, Colo.
W. H. Lytle, Salem, Ore.
W. A. Hagan, 320 The Parkway, Ithaca, N. Y.
C. F. Schlotthauer, Mayo Foundation, Rochester, Minn.
R. O. Biltz, Box 327, Harrisburg, Pa.

Committee on Resolutions

B. T. Simms, *Chairman*, Ore. Agr. College, Corvallis, Ore.
E. E. Wegner, College Station, Pullman, Wash.
J. B. Lentz, 3 Dana St., Amherst, Mass.
W. T. Spencer, 1250 N. 37th St., Lincoln, Nebr.
R. P. Marsteller, College Station, Texas.

Committee on Policy

W. H. Welch, *Chairman*, Lexington, Ill.
*M. Jacob, Box 416, Knoxville, Tenn.
*H. Preston Hoskins, 716 Book Bldg., Detroit, Mich.
*C. H. Stange, Iowa State College, Ames, Iowa.
*T. H. Ferguson, 421 Broad St., Lake Geneva, Wis.

Committee on Veterinary Biological Products

H. J. Shore, *Chairman*, Fort Dodge Serum Co., Fort Dodge, Iowa	1931
E. R. Steel, 8023 Wornall Road, Kansas City, Mo.	1930
F. A. Imler, 19 Federal Bldg., Kansas City, Kans.	1932
M. J. Harkins, 110 East 10th Ave., Conshohocken, Pa.	1933
B. J. Killham, 721 State Office Bldg., Lansing, Mich.	1934

Committee on Proprietary Pharmaceuticals

H. D. Bergman, *Chairman*, Ames, Iowa	1933
G. H. Glover, Fort Collins, Colo.	1930
R. S. Amadon, 39th St. & Woodland Ave., Philadelphia, Pa.	1931
R. A. Craig, 621 Waldron St., West Lafayette, Ind.	1932
E. L. Quitman, 1514 W. Van Buren St., Chicago, Ill.	1934

Committee on History

J. P. Foster, *Chairman*, 4053 Bryant Ave. S., Minneapolis, Minn.
P. A. Fish, 931 E. State St., Ithaca, N. Y.
C. J. Marshall, 5031 Pine St., Philadelphia, Pa.
D. S. White, 1490 Cardiff Rd., Upper Arlington, Columbus, Ohio.
C. D. McGilvray, Ontario Veterinary College, Guelph, Ont.

Committee on Tuberculosis

C. E. Cotton, *Chairman*, 3145 Portland Ave., Minneapolis, Minn.
J. S. Healy, 110 E. Washington Ave., Madison, Wis.
E. A. Watson, Animal Diseases Institute, Ottawa, Ont., Canada.
A. F. Schalk, State College Station, Fargo, N. Dak.
C. H. Case, 26 Orchard Road, Akron, Ohio.

Committee on Abortion

C. P. Fitch, *Chairman*, University Farm, St. Paul, Minn.
M. F. Barnes, Box 403, Harrisburg, Pa.
Herbert Lothe, 920 Barston St., Waukesha, Wis.
W. E. Cotton, B. A. I. Exp. Sta., Bethesda, Md.
E. T. Hallman, 615 Sunset Lane, East Lansing, Mich.

*Ex officio

COMMITTEE ON DISTEMPER

J. V. Lacroix, *Chairman*, 1817 Church St., Evanston, Ill.
F. H. Miller, 300 Webster Ave., New Rochelle, N. Y.
John Reichel, Glenolden, Pa.
T. H. Agnew, 80 N. Daisy St., Pasadena, Cal.
O. V. Brumley, Ohio State University, Columbus, Ohio.

COMMITTEE ON PREVENTION OF TRANSMISSIBLE DISEASES OF ANIMALS

Jacob Traum, *Chairman*, 101 Budd Hall, Univ. of Cal., Berkeley, Cal.
W. W. Dimock, University of Kentucky, Lexington, Ky.
E. W. Price, Zoological Div., B. A. I., Washington, D. C.
Alvin Broerman, State Serum Institute, Reynoldsburg, Ohio.
J. F. Shigley, State Agr. College, State College, Pa.
F. B. Hadley, University of Wisconsin, Madison, Wis.

COMMITTEE ON INTERNATIONAL VETERINARY CONGRESS

J. R. Mohler, *Chairman*, B. A. I., Washington, D. C.
Adolph Eichhorn, c/o Lederle Antitoxin Labs., Pearl River, N. Y.
V. A. Moore, N. Y. State Veterinary College, Ithaca, N. Y.
George Hilton, 126 Lewis St., Ottawa, Ont.
D. S. White, 1490 Cardiff Rd., Upper Arlington, Columbus, Ohio.
L. A. Merillat, 569 W. Van Buren St., Chicago, Ill.
C. J. Marshall, 5031 Pine St., Philadelphia, Pa.
C. A. Cary, Alabama Polytechnic Institute, Auburn, Ala.
J. W. Connaway, Univ. of Missouri, Columbia, Mo.
L. Van Es, University Farm, Lincoln, Nebr.
R. R. Dykstra, Kansas State Agr. College, Manhattan, Kans.
C. M. Haring, 2405 Hillside Ave., Berkeley, Cal.

COMMITTEE ON WATER-BORNE DISEASES OF ANIMALS

L. W. Goss, *Chairman*, Ohio State University, Columbus, Ohio.
W. L. Boyd, University Farm, St. Paul, Minn.
L. T. Giltner, Bureau of Animal Industry, Washington, D. C.
Chas. Murray, Iowa State College, Ames, Iowa.

COMMITTEE ON STANDARD MILK CONTROL CODE

George W. Grim, *Chairman*, Township Bldg., Ardmore, Pa.
E. D. King, Jr., Valdosta, Ga.
John P. Turner, 1357 Kennedy St. N. W., Washington, D. C.
John B. Hollingsworth, 105 Cambridge St., Ottawa, Ont., Canada.
Roy F. Leslie, 127 City Hall, Cleveland, Ohio.

COMMITTEE ON HUMANE SOCIETY HOSPITALS

R. S. MacKellar, *Chairman*, 351 W. 11th St., New York, N. Y.
W. G. Hollingworth, Apt. 31, 1514 Genesee St., Utica, N. Y.
H. K. Miller, 151 Harrison Ave., Mamaroneck, N. Y.

COMMITTEE ON POULTRY DISEASES

H. W. Graybill, *Chairman*, 1021 Daisy Ave., Long Beach, Calif.
B. A. Beach, Univ. of Wisconsin, Madison, Wis.
E. L. Stubbs, 39th St. & Woodland Ave., Philadelphia, Pa.
Iva Dunn, Atkins, Iowa.
Robert Graham, Univ. of Illinois, Urbana, Ill.

COMMITTEE ON PROGRAM FOR A. A. A. S. MEETING

Ward Giltner, *Chairman*, Box 955, East Lansing, Mich.
C. H. Stange, Iowa State College, Ames, Iowa.
C. P. Fitch, University Farm, St. Paul, Minn.

DELEGATE TO FOURTH WORLD'S POULTRY CONGRESS, LONDON, 1930

W. T. Johnson, Corvallis, Ore.

REPRESENTATIVE TO NATIONAL POULTRY SANITATION COMMISSION

N. S. Mayo, c/o Abbott Labs., North Chicago, Ill.

Representative to National Research Council

W. A. Hagan, 320 The Parkway, Ithaca, N. Y.

Representative on Board of Managers, Horse Association of America

T. A. Sigler, Greencastle, Ind.

Representative to the Amepican Association fop the Advancement of Science

Ward Giltner, Box 955, East Lansing, Mich.

Resident State Secretaries

Alabama	D. J. Meador, c/o L. & N. R. R., Montgomery.
Arizona	M. Shipley, Box 311, Phoenix.
Arkansas	J. H. Box, Old State House, Little Rock.
California	W. L. Curtis, 1264 W. 2nd St., Los Angeles.
Colorado	I. E. Newsom, Colo. Agr. College, Fort Collins.
Connecticut	Edwin Laitinen, 993 N. Main St., West Hartford.
Delaware	C. C. Palmer, Univ. of Delaware, Newark.
Dist. of Columbia	F. W. Miller, 1001 Irving St. N. E., Washington.
Florida	T. J. Mahaffy, 816 Broad St., Jacksonville.
Georgia	E. D. King, Jr., Valdosta.
Idaho	W. A. Sullivan, Box 809, Boise.
Illinois	C. C. Hastings, Williamsville.
Indiana	W. J. Armour, 114 S. Third St., Goshen.
Iowa	John B. Bryant, Mt. Vernon.
Kansas	B. W. Conrad, Sabetha.
Kentucky	C. G. Kreidler, Maysville.
Louisiana	Henry H. Baur, Box 248, Monroe.
Maine	M. E. Maddocks, 7 School St., Augusta.
Maryland	E. M. Pickens, University of Maryland, College Park.
Massachusetts	H. W. Jakeman, 44 Bromfield St., Boston.
Michigan	A. S. Schlingman, c/o Parke, Davis & Co., Detroit.
Minnesota	H. C. H. Kernkamp, University Farm, St. Paul.
Mississippi	Hartwell Robbins, 605 Millsaps Bldg., Jackson.
Missouri	J. D. Ray, 400 New Centre Bldg., Kansas City.
Montana	Hadleigh Marsh, Livestock Sanitary Board, Helena.
Nebraska	Bernhard Witt, Scribner.
Nevada	Edward Records, University of Nevada, Reno.
New Hampshire	A. L. Edmunds, Franklin.
New Jersey	J. G. Hardenbergh, Walker-Gordon Laboratory Co., Plainsboro.
New Mexico	F. H. Barr, 112 S. Broadway, Albuquerque.
New York	J. E. Crawford, Far Rockaway, L. I.
North Carolina	William Moore, N. C. Dept. of Agr., Raleigh.
North Dakota	A. F. Schalk, State College Sta., Fargo.
Ohio	C. McCandless, 96 S. Ardmore Rd., Bexley.
Oklahoma	C. R. Walter, 538 S. Madison, Tulsa.
Oregon	T. B. Carter, Gresham.
Pennsylvania	Freeman A. Marshall, 498 S. Fifth St., Indiana.
Rhode Island	J. S. Barber, 14 Washington St., Central Falls.
South Carolina	M. R. Blackstock, 157 Hampton Ave., Spartanburg.
South Dakota	G. P. McCue, 115 Fourth Ave. S. W., Aberdeen.
Tennessee	James M. Jones, Lewisburg.
Texas	N. F. Williams, 701 Wheat Bldg., Fort Worth.
Utah	H. J. Frederick, College Hill, Logan.
Vermont	L. H. Adams, State House, Montpelier.
Virginia	H. C. Givens, 1102 State Office Bldg., Richmond.
Washington	John E. McCoy, Box 136, Pullman.
West Virginia	S. E. Hershey, Box 283, Charleston.
Wisconsin	Chas. F. Van de Sand, Kiel.
Wyoming	Cecil Elder, University of Wyoming, Laramie.

RESIDENT TERRITORIAL SECRETARIES

Alaska	E. F. Graves, c/o Office of the Governor, Box G, Juneau
Canal Zone	T. L. Casserly, Drawer "K," Cristobal.
Haiti	I. B. Boughton, Service Technique, Port au Prince.
Hawaii	L. E. Case, Box 295, Honolulu.
Philippine Islands	S. Youngberg, Bureau of Agriculture, Manila.
Porto Rico	H. L. Van Volkenberg, Agr. Exp. Sta., Mayaguez.
Virgin Islands	G. A. Roberts, Christiansted, St. Croix.

RESIDENT PROVINCIAL SECRETARIES

Alberta	J. C. Hargrave, Medicine Hat.
British Columbia	J. G. Jervis, Milner.
Manitoba	Alfred Savage, Manitoba Agri. College, Winnipeg.
New Brunswick	Adam T. McLean, W. Market St., Moncton.
Nova Scotia	J. Steen, Customs Bldg., Halifax.
Ontario	R. A. McIntosh, Ont. Vet. College, Guelph.
Prince Edw. Isl'd	W. H. Pethick, Charlottetown.
Quebec	A. A. Etienne, 1125 Drummond St., Montreal.
Saskatchewan	R. G. Chasmar, Hanley.

FOREIGN CORRESPONDING SECRETARIES

Argentina	Lorenzo P. Garrahan, Florida 668, Buenos Aires.
Colombia	Roberto P. Guerrero, Apartado 149, Bogota.
Cuba	L. A. Beltran, Castillo de la Fuerga, Box 883, Havana.
Egypt	J. E. Aghion Bey, 6 Midan Soliman Pacha, Cairo.
England	R. W. Tuck, c/o American Consulate, 18 Cavendish Sq., London, W. 1.
Hungary	A. Kotlan, Royal Hungarian Vet. College, Budapest VII.
Jamaica	Stephen Lockett, Dept. of Agr., Kingston.
Mexico	L. Santa Maria, Apartado Postal 2067, Mexico, D. F.
Peru	J. F. Mitchell, c/o Cerro de Pasco Copper Corp., Oroya.
St. Kitts	E. F. Jardine, Box 18, Basseterre.
Scotland	A. W. Whitehouse, Glasgow Veterinary College, 83 Buccleuch St., Glasgow.

AMERICAN VETERINARY MEDICAL ASSOCIATION

Minutes of Special Meeting of Executive Board

A special meeting of the Executive Board was held in Room 104, La Salle Hotel, Chicago, Ill., Wednesday evening, December 4, 1929, at 8:00 p. m. Board members present were:

Dr. C. H. Stange, Chairman, District No. 5.
Dr. George Hilton, District No. 1.
Dr. E. P. Althouse, District No. 2.
Dr. L. A. Merillat, District No. 3.
Dr. C. A. Cary, District No. 4.

Absent:

Dr. Geo. H. Hart, District No. 6.
Dr. R. S. MacKellar, Member-at-Large.

Also present were Dr. M. Jacob, Treasurer, Dr. T. H. Ferguson, President, and Dr. H. Preston Hoskins, Secretary-Editor.

The first item of business was the selection of dates for the 1930 convention in Los Angeles. In this connection Secretary

Hoskins read a communication from Dr. W. L. Curtis, secretary of the Southern California Veterinary Medical Association, recommending either the first or second week in September. Considerable discussion followed and several members of the Board registered objections to holding the meeting at such a late date. A motion was made by Dr. Cary and seconded by Dr. Hilton that the 1930 convention be held during the third week in August (August 19-20-21-22), provided that these dates are acceptable to the Los Angeles veterinarians. The motion was carried unanimously.

Secretary Hoskins then stated that he had received a joint proposition from the Chicago Northwestern and Union Pacific Railroads in connection with the official route to Los Angeles. In return for being designated as the official route to the convention, these lines would take four pages of advertising in the JOURNAL at the regular rate. It was moved by Dr. Cary and seconded by Dr. Merillat that the Secretary be authorized to accept this proposal. The motion was carried unanimously.

The next item of business was to act upon several applications for membership that had been referred to the Executive Board. The first was that of Dr. George F. Jungerman, of Hiawatha, Kans. The Secretary reported that, following the first listing of the application of Dr. Jungerman in the November issue of the JOURNAL, he received a number of letters from members registering objections to the admission of Dr. Jungerman as a member of the A. V. M. A. After a very thorough discussion of this case, Dr. Hilton moved that the application of Dr. Jungerman be rejected. The motion was seconded by Dr. Althouse and carried unanimously.

The application of Dr. Edgar B. Dibbell, of Baltimore, Md., was then presented under Section 2 of Article 3 of the By-Laws. Dr. Dibbell is a graduate of the United States College of Veterinary Surgeons, class of 1921, the institution not having been on the accredited list at the time of his graduation. Dr. Hilton moved that the application be approved. Motion was seconded by Dr. Althouse and unanimously carried.

Secretary Hoskins then asked for instructions relative to the publication of a new edition of the A. V. M. A. Membership Directory. He pointed out that the unusually large number of members admitted during the past year made the publication of another membership directory rather imperative. Dr. Cary moved that a new edition be published as soon as possible after

the first of the year. The motion was seconded by Dr. Merillat and carried unanimously.

Attention was then given to the several amendments which had been introduced at the Detroit meeting. The amendment first considered was as follows: Article V, Section 2, of the Constitution now reads:

> The President shall preside at all meetings of the Association and shall deliver an address at the regular annual meeting. He shall appoint all committees and shall be ex officio a member of each standing committee. He shall appoint those officers whose appointments are not otherwise provided for and perform such duties as properly devolve upon a president. No president shall be eligible for re-election to that office.

Change to read:

> The President shall preside at all meetings of the Association and shall deliver an address at the regular annual meeting. He shall appoint all committees and shall be ex officio a member of the Executive Board and of each standing committee. He shall appoint those officers whose appointments are not otherwise provided for and perform such duties as properly devolve upon a president. No president shall be eligible to re-election to that office.

It was moved by Dr. Cary and seconded by Dr. Hilton that the Executive Board approve this amendment. Carried unanimously.

The second amendment was in connection with Article V, Section 7, of the Constitution, which now reads as follows:

> There shall be an executive board consisting of one member-at-large and one member from each Executive Board district that shall be created as hereinafter provided. (By-laws, Article 10.)

Change to read:

> There shall be an executive board consisting of one member-at-large and one member from each Executive Board district that shall be created as hereinafter provided, the immediate past president and the president (ex officio.) (By-laws, Article 10.)

It was moved by Dr. Hilton, seconded by Dr. Althouse, that the Executive Board approve this amendment. Carried unanimously.

The third amendment to be considered was that in connection with Article 5, Section 1, of the By-laws, which now reads as follows:

> There shall be a membership fee of five dollars ($5.00). The annual dues of active members, including subscription to the JOURNAL, shall be five dollars ($5.00), payable in advance on the first day of January.

Change to read:

> There shall be a membership fee of eight dollars ($8.00). The annual dues of active members, including subscription to the JOURNAL, shall be eight dollars ($8.00), payable in advance on the first day of January.

Considerable discussion took place relative to the advisability, or otherwise, of increasing the membership fee and annual dues. No action was taken.

The fourth amendment to be considered was that in connection with Article 12, Section 1, of the By-laws, which now reads as follows:

The Association shall be divided into the following sections:
A. General Practice.
B. Sanitary Science and Food Hygiene.
C. Education and Research.
D. Small Animals.
E. Poultry.
The Executive Board may make additional sections as deemed expedient.

Change to read:

The Association shall be divided into the following sections:
A. General Practice.
B. Disease Control and Food Hygiene.
C. Experimental Pathology.
D. Small Animals.
E. Poultry.
The Executive Board may make additional sections as deemed expedient.

It was moved by Dr. Hilton and seconded by Dr. Merillat that the Executive Board recommend to the Association that the present Section on Education and Research be designated as the Section on Research. Unanimously carried.

It was moved by Dr. Cary and seconded by Dr. Hilton that the Executive Board recommend that no change be made in the name of the present Section on Sanitary Science and Food Hygiene. Carried unanimously.

The next amendment was not introduced at Detroit, but will be introduced at Los Angeles, in 1930. The amendment is recommended by the Executive Board as the outcome of a similar recommendation made by the Special Committee on Publicity and is as follows:

Amend Article 17—Standing Committees, Section 1. Add line eleven to read as follows: 9. Public Relations.

Add Section 11 (present Section 10 revised)—The President shall appoint the chairman of each committee provided in Sections 3, 4, 6, 7, 8, 9 and 10. In the case of the Committee on Veterinary Biologics, the Committee on Proprietary Pharmaceuticals, the Committee on Education, the Committee on Legislation and the Committee on Public Relations, the chairman shall be appointed from the hold-over members.

Amend Section 10 (new) to read as follows: The Committee on Public Relations shall consist of five members, to be appointed by the President, each to serve for five years, provided, however, that the first Committee to be appointed under this Section shall serve for the following terms: one member for one year, one for two years, one for three years, one for four years and one for five years.

Chairman Stange reported that he had appointed the following special committee to work out the details of selecting office quarters in Chicago: Drs. L. A. Merillat, Evanston, Ill., *Chairman*,

W. H. Welch, Lexington, Ill., and H. Preston Hoskins, Detroit, Mich. The Board gave this special committee power to act.

The Secretary then reported that he had received several complaints to the effect that, in view of the fact that the A. V. M. A. Committee on Abortion, in the report made at the 1929 meeting in Detroit, had unqualifiedly condemned the use of living organism abortion vaccines, and the Association had accepted the report, it was inconsistent upon the part of the Association to admit advertisements of such products to the advertising pages of the JOURNAL. After considerable discussion, Dr. Cary moved that advertisements of living organism abortion vaccines be excluded from the JOURNAL. The motion was seconded by Dr. Hilton and carried.

The Secretary then advised the Board that it was becoming an increasingly difficult task to find the necessary space for publishing all of the material submitted to the JOURNAL. He pointed out that, with the increase in the number of sections in the A. V. M. A., the number of papers presented at our annual meetings was steadily increasing. Authorization was asked to increase the size of the JOURNAL approximately 16 pages per month at an estimated cost of $100 each issue. It was moved by Dr. Cary that this authorization be given. Motion was seconded by Dr. Hilton and carried.

The meeting adjourned at 11:30 p. m.

H. PRESTON HOSKINS, *Secretary.*

Bibliography on Bovine Infectious Abortion for 1928

The bibliography on bovine infectious abortion for 1927, published in the JOURNAL, August, 1929, was prefaced with a statement as to where one might find previous bibliographies. The further publication of the annual increments to literature on abortion has become a burden that no professional or scientific journal can be asked to assume. Therefore, mimeographed copies of the bibliography on bovine infectious abortion for 1928 have been prepared and will be mailed upon receipt of request addressed to the Bacteriological Laboratory, Michigan State College, East Lansing, Mich.

W. G.

Have you written up that case report that you promised? Your colleagues would probably be as interested in it as you were.

ASSOCIATION MEETINGS

INTERNATIONAL ASSOCIATION OF DAIRY AND MILK INSPECTORS

This association held its eighteenth annual convention at the Hotel Peabody, Memphis, Tenn., October 7-8-9, 1929. Over two hundred members and guests were in attendance, there being a considerable number of veterinarians present in their official capacities.

Twenty-two papers and thirteen comprehensive committee reports were considered in six morning, afternoon and evening sessions. In his paper on "Fundamentals of Dairy Inspection," Prof. A. D. Burke, Department of Dairying, Alabama Polytechnic Institute, stated that the milk inspector of the future might have to be a combination of veterinarian and dairyman, well grounded in bacteriological, chemical and dairy science and in engineering technic. Such requirements may have a bearing eventually on college curricula. Prof. Burke defined the successful inspector as one who "educates the ignorant, eliminates the dishonest and protects the progressive."

In a paper on " 'Safety First' in Milk Sanitation," Dr. Paul B. Brooks, Deputy Commissioner of Health of New York State, took recognition of the fact that universal pasteurization is a long way in the future and that, in the meantime, suitable measures must be adopted for the protection of raw milk supplies. Dr. Brooks' safety measures contemplate three principal phases: protection against mastitis, protection against human cases of communicable disease, and protection against carriers. The first he would approach by requiring manual examination of the udder of each cow daily by the dairyman; by foremilk examination; by exclusion of milk from any cow showing mastitis; by continuous veterinary inspection (at public expense); and by weekly bacteria counts on herd samples (also at public expense). Protection against human types of infection would be gained by the immediate reporting of all cases of illness by the dairyman, however trivial. Protection against carriers would require typhoid history and Widal test of all milk handlers, with a sufficient number of samples of excreta for laboratory examination to give reasonable assurance of freedom from carrier condition; also, throat swab

initially and later, when any sign of sore throat is shown by a milk handler.

A number of veterinarians contributed to the program. Dr. W. G. Hollingworth, of Utica, New York, gave one of his characteristic presentations in "The Pitfalls Connected with Dairy and Milk Inspection." The paper was very comprehensive, loaded with good common sense and enlivened by the Doctor's usual wit and humor. Dr. Alexander F. Eagle, Dairy Veterinarian of San Francisco, upheld the traditions of California and his associates by his discussion on "San Francisco's Milk Supply and Its Problems," in that he showed the milk supply of his city to be better than that encountered in most sections of the country. From the discussion that followed Dr. Eagle's paper, it would seem that San Francisco has no milk problems compared to other cities.

Dr. F. D. Holford, Chief Veterinarian of the Borden's Farm Products Company of New York City, spoke on "The Relation of Udder Infection to Human Health." Dr. Holford's long experience in dairy inspection work enables him to speak with authority on any phase of milk control work. In a symposium on certified milk, there were three papers. One of these was by Dr. C. I. Corbin, Sheffield Farms Company, New York City, on "Progress in the Control of the Production of Certified Milk." Dr. Corbin reviewed the improvements that have been made in technical procedures for the control of high-quality milk supplies. Dr. J. G. Hardenbergh, of the Walker-Gordon Laboratory Company, Inc., Plainsboro, N. J., spoke on "Laboratory Control of Certified Milk on the Farm" and detailed the system used on the larger certified milk farms in checking the purity and safety of the milk produced.

A significant feature of the meeting was the attitude expressed on the subject of human infection with organisms of the abortus-melitensis group. Although there was a great deal of discussion from a number of angles, the consensus of opinion of lay and professional men, both medical and veterinary, who are actively engaged in official milk inspection work, was that present knowledge of the subject does not warrant any radical action toward milk as a carrier of the infection. The sensational and unscientific treatment which has been accorded the subject in certain sections of the country and in certain lay publications was held to be unwarranted.

J. G. HARDENBERGH, *Reporter*

KANSAS CITY ASSOCIATION OF VETERINARIANS

August Meeting

The Kansas City Association of Veterinarians held the regular August meeting in the form of a basket picnic at Fairyland Park, August 20, 1929. About thirty-five members and friends were present, and enjoyed a pleasant evening, following the dinner, doing the park in its lighted glory.

September Meeting

The September meeting was devoted to a round-table discussion of various problems and case reports from practitioners. Several members of the Kansas City Association of Veterinarians attended the Detroit Convention of the A. V. M. A. and reported things of special interest they had been privileged to see and hear at this meeting.

October Meeting

The regular October meeting was held at the Baltimore Hotel, in conjunction with the local Association of Bureau of Animal Industry Veterinarians. Dr. George W. Stiles, of Denver, Colo., favored the Association with a detailed description of anaplasmosis in cattle. The entire program was devoted to this subject. Seventy-five veterinarians from in and around Kansas City were in attendance.

November Meeting

The regular November meeting of the Association was held November 12, 1929, at the Baltimore Hotel. The program for the evening was devoted to the problems of sheep practice. Dr. A. T. Kinsley, of Kansas City, conducted a round-table on sheep diseases and Dr. W. H. Bailey, of St. Joseph, presented an interesting paper and a number of good specimens from diseased sheep. Much interest was shown in the discussion. Thirty-five veterinarians were present.

J. D. Ray, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held at the Academy of Medicine Building, 103rd Street and Fifth Avenue, New York City, November 6, 1929. The meeting was called to order by President H. K. Miller.

Dr. C. H. Higgins, of the Lederle Antitoxin Laboratories, gave a very interesting and thoroughly instructive talk on distemper and the preventive measures necessary to control it. He discussed thoroughly the method which Laidlaw and Dunkin have developed. He brought out the necessity of ascertaining whether or not dogs have been exposed to distemper prior to vaccination, as the dogs were apt to develop distemper if they have been exposed and then inoculated with the vaccine.

A general discussion followed and many questions were asked Dr. Higgins, who patiently explained the technic and the modus operandi of injecting the vaccine and virus. Drs. Little, Ticehurst, Crawford, Rohrer, Goodman, Miller and several others engaged in the discussion and many interesting points were brought out.

A rising vote of thanks was extended to Dr. Higgins for his kindness in discussing such an important and controversial question.

Dr. R. S. MacKellar stated that the Code of Ethics of the American Humane Association had been adopted by the American Veterinary Medical Association at the recent meeting in Detroit.

There being no further business, motion made and seconded that the meeting be adjourned.

RAYMOND J. GARBUTT, *Secretary*

SOUTHWEST MISSOURI VETERINARY ASSOCIATION

The Southwest Missouri Veterinary Association held its fall meeting at the Y. W. C. A., at Joplin, November 13, 1929. A very interesting and elaborate program had been arranged for the occasion.

"Veterinary Dietetics," by Dr. W. O. Hodge, of Brookline Station, was followed by a good discussion and many points of interest to practicing veterinarians in that territory were brought out.

"Anaplasmosis: Diagnosis and Treatment," by Dr. C. D. Meredith, of Joplin, also was a feature subject of the meeting. Practically every veterinarian in attendance had something to say on this subject. This disease was very prevalent in the territory around Joplin the past year.

"Prevention and Control of Abortion," by Dr. D. B. Morgan, of Neosho, brought forth the usual array of ideas relative to this disease. "Sterility in Cattle," by Dr. W. J. Houser, of Carthage,

was discussed from the various causative features that were considered to be associated with this disease. "Is Canine Distemper a Danger to the Human?" by Dr. W. J. Stone, of Joplin, was a relation of personal experience and observations.

"Round Table on Old Fashioned Drugs," by Dr. C. D. Folse, of Kansas City, brought forth many interesting features and was handled in a manner to impress the various practitioners present with the fact that they were depending too much on pharmaceutical preparations sold by the various commercial concerns and stressed the fact that every veterinarian should brush up on his materia medica.

Twenty-three veterinarians from southwest Missouri and adjoining states were in attendance. Most of them were accompanied by their wives, who were entertained by the wives of the local veterinarians. It was decided to hold the spring meeting in Springfield, Mo.

J. D. RAY, *Reporter.*

HUDSON VALLEY VETERINARY MEDICAL SOCIETY

The regular quarterly meeting of the Hudson Valley Veterinary Medical Society was held at the Nelson House, Poughkeepsie, N. Y., November 13, 1929. President Wm. Henry Kelly presided. There were thirty-four veterinarians present.

This being the annual meeting, the report of the Secretary-Treasurer was received and accepted. The election of officers followed, Dr. I. O. Denman, of Middletown, being elected president and Dr. A. Eichhorn, of Pearl River, vice-president. The Secretary-Treasurer was re-elected. Censors elected were: Dr. R. H. Spaulding, White Plains; Dr. George L. Stringham, Wappingers Falls; Dr. Albert N. Towner, Towners; Dr. R. S. Banks, Rhinebeck; and Dr. H. C. Parker, Hudson.

Among the visitors present were Drs. A. K. Zellner, of Oneida, and Don A. Boardman, of Rome, who discussed the question of an organization to protect the interests of the accredited veterinarians of the State, who, under the so-called Kirkland Law, will be called upon to tuberculin-test accredited herds at state expense. After considerable discussion the President-elect was authorized to appoint a committee to call a meeting of accredited veterinarians of eastern New York to consider the formation of such an organization.

Dr. L. L. Parker, of Catskill, then discussed "The Treatment of Scours in Calves." He recommended the use of oatmeal water

and lime water as an efficient method of reducing losses, stating that in his opinion the disease was due largely to feeding errors.

Dr. R. H. Spaulding, of White Plains, then outlined his experiences in the treatment of distemper in dogs by the Laidlaw-Dunkin method. He reported a number of apparent "breaks" following the double treatment. Investigation was made under the supervision of Dr. Eichhorn, who, in the discussion, advised that he did not feel that the evidence was sufficient to show definitely the treatment to be ineffective and in his opinion other complications were the cause of the unsatisfactory outcome following vaccination. Various points in relation to treatment of distemper by this method were brought out. Dr. Spaulding stated that in some cases a temperature rise had followed the administration of vaccine and, by withholding virus, the patient had recovered without further complications.

Dr. George A. Knapp, of Millbrook, discussed mastitis and stated that he obtained best results by using a mixed bacterin, following this with equal parts of formalin and turpentine, two ounces, given in capsules by the mouth, once a day for three days. Dr. Knapp also referred to the use of acriflavin and Dr. A. N. Towner reported the use of a German apparatus for determining the presence of mastitis before visible symptoms occurred.

Dr. A. L. Smith, of Mechanicsville, referred to a proposal by the Department of Education that the profession approve of an increase in the annual registration fee from one to two dollars, the fund to be used in the investigation of illegal practitioners. This was left to the Legislative Committee for action.

J. G. WILLS, *Secretary.*

BAY COUNTIES VETERINARY MEDICAL ASSOCIATION

The annual meeting-banquet of the Bay Counties (Calif.) Veterinary Medical Association was held at the Elk's Club, San Francisco, Calif., Tuesday, November 13, 1929, and the following officers were elected for the ensuing year: President, Dr. Joseph M. Arburua; vice-president, Dr. Oscar J. Kron; secretary-treasurer, Dr. Geo. M. Simmons.

MICHIGAN-OHIO VETERINARY MEDICAL ASSOCIATION

A meeting of the Michigan-Ohio Veterinary Medical Association was held at the Court House, Adrian, Mich., November 20,

1929. There were twenty-two veterinarians from Michigan and Ohio in attendance.

The meeting was opened by Dr. C. H. Hoffmire, President, of Adrian, with a few remarks. A talk, entitled, "Retained Placentas in Cows" was presented by Dr. H. J. Seaman, of Wauseon, Ohio.

Dr. E. T. Hallman, of Michigan State College, gave a most interesting talk on contagious abortion. Vaccination of cattle against this disease and the findings on nine Commercial abortion vaccines which were tested at the Michigan State College. He advised the practicing veterinarian to get ready to conduct the rapid agglutination test when the call comes from the client.

Dr. B. J. Killham, State Veterinarian, gave some very interesting facts concerning the control of contagious diseases in the state of Michigan. Dr. H. J. Stafseth, of Michigan State College, gave a very interesting talk on chicken-pox and roup. He brought out the fact that these two conditions are separate and distinct diseases.

Dr. S. G. Colby, of Monroe, Mich., gave some recent observations on hog cholera vaccination, especially pertaining to vaccination of pregnant sows and small pigs. Dr. Ward Giltner, dean of the Veterinary Division, Michigan State College, and Dr. H. Preston Hoskins, Secretary-Editor, American Veterinary Medical Association, were there to help with the meeting. They brought out many interesting points in the discussion of the talks.

Following an adjournment of the meeting at the Court House, a banquet was held at the Powers Hotel at six o'clock.

E. C. W. SCHUBEL, *Secretary*.

CENTRAL NEW YORK VETERINARY MEDICAL ASSOCIATION

The twentieth semi-annual meeting of the Central New York Veterinary Medical Association was held at the Onondaga Hotel, Syracuse, November 20, 1929. The meeting was called to order at 2:30 p. m. by Dr. A. K. Zellner, President. Roll call showed twenty-five members in attendance with two visitors.

After some routine business was transacted, a motion prevailed that the Secretary write the proper authorities at Washington asking why veterinarians in the Albany and Buffalo districts are allowed only two gallons of alcohol per year, when in some other districts veterinarians are able to procure as high as five gallons per year.

Dr. W. F. Burleigh, of Oriskany Falls, read a very interesting paper, entitled, "Treatment of Foot-rot in Cattle." Dr. E. L. Monfort, of Richfield Springs, presented a paper, entitled, "Veterinary Science as Taught in the New York State School of Agriculture at Morrisville, N. Y."

Dr. D. M. Hoyt, of Canastota, gave a very interesting demonstration of the rapid agglutination test for abortion, which was very much appreciated by all present. Dr. C. R. Baldwin, of Fulton, rendered a report on his trip to Albany in behalf of the accredited veterinarians of New York State. Dr. Baldwin stated that he was received in a very friendly spirit at Albany and added that the authorities would be glad to cooperate with the veterinarians. Dr. D. A. Boardman, of Rome, reported on his visit to the Hudson Valley Veterinary Association in the interests of the accredited veterinarians of New York State. He requested that delegates from the central and western parts of the State be present at the next meeting.

Dr. Karl B. Hanson, of the U. S. Experimental Fur Farm, Saratoga Springs, gave a very interesting talk on fox farming as carried on at the farm. His talk was followed with a number of moving-picture films showing the proper way of handling foxes. This subject was very interesting and highly appreciated by everybody.

W. B. SWITZER, *Secretary.*

HORSE ASSOCIATION OF AMERICA

The tenth annual meeting of the Horse Association of America was held in Chicago, Ill., December 4, 1929, and was the best meeting in the history of the organization. Addresses were made covering every branch of horse and mule production and use, in which striking evidence was shown of a very definite advance. The details will be given in the annual report of the Association.

It was pointed out by speakers that good draft horses or mules may be raised to three years of age for $90 a head; that a mature work animal on farms may be kept for $60 a year; and that any good farmer south of the 42nd degree of latitude and east of the 100th meridian of longitude can achieve this by adopting methods described in Leaflet 195, "Keeping Farm Teams at Low Cost."

A thing which made an instant hit at the International Live Stock Show was our exhibit of models of big teams, presented in one of the booths of the U. S. Department of Agriculture, and also at our banquet. Horses, harnesses and implements were made

one-seventh actual size into five- and six-horse outfits on gang plows and an eight-horse outfit on a tandem disc. Men of long experience in the big-hitch work declare the models are the greatest help ever offered for teaching the work, and the Department of Agriculture rated them the most interesting and effective exhibit in its entire show.

Farmers and business men alike are increasingly cognizant that horses and mules are fundamental to low costs and to a proper distribution of labor and of markets. The whole situation, we feel, is distinctly better.

WAYNE DINSMORE, *Secretary.*

A Desirable Move

Creation of a state department of animal industry was asked by the Illinois State Veterinary Medical Association, which met in Chicago early in December. Veterinarians will make an appeal for legislation to secure this department. The fact that the work of such a department is now directed by a layman, under supervision of the State Department of Agriculture, which has charge of all live stock and disease control, was pointed out as one of the reasons for making the change.

Main Building of Sero-Therapeutic Institute, Milan, Italy.

NECROLOGY

WILLIAM T. KING

Dr. William T. King, of Quenemo, Kansas, died September 26, 1929, of heart disease. He was a graduate of the Kansas City Veterinary College, class of 1895, and practiced at Olathe, Kansas, until about twelve years ago, when he removed to Quenemo, where he engaged in farming and continued to practice to a limited extent.

L. L. WHITNEY

Dr. L. L. Whitney, of Lyndon, Kansas, died September 27, 1929, of diabetes. He was a graduate of the Kansas City Veterinary College, class of 1918, and was engaged in general practice.

VALENTIN SCHAEFER

Dr. Valentin Schaefer, of Tekamah, Nebraska, died at his home, November 15, 1929, at the age of 81. He was a graduate of the Chicago Veterinary College, class of 1891, and was the oldest practitioner in the state of Nebraska. He was associated in practice with his son, Dr. George Schaefer (K. C. V. C. '13).

The deceased joined the A. V. M. A. in 1898.

WILLIAM P. BROWER

Dr. William P. Brower, of Menno, S. D., was drowned in the James River, near Menno, November 27, 1929. According to reports, Dr. Brower was returning home from a professional call and stopped at the James River, where he had sighted some ducks. He crossed the river on the ice and shot two ducks. On the way back to his car he broke through the ice and sank in fifteen feet of water. His body was recovered four hours later.

Dr. Bower was a graduate of Iowa State College, class of 1923, and practiced at Kanawha, Iowa, immediately following graduation. Later he located at Menno, S. D., and built up a fine practice there. He joined the A. V. M. A. in 1923. At the 1929 meeting of the South Dakota Veterinary Association he was elected Secretary-Treasurer. He is survived by his widow and an infant daughter.

EDWARD E. TERRY

Dr. Edward E. Terry, of Holmesburg, Philadelphia, Pa., died on November 28, 1929, of angina pectoris. He was born at Trevose, Bucks County, Pa., January 1, 1863, and was a graduate of the University of Pennsylvania, class of 1893. He practiced at Holmesburg continuously from the time of his graduation.

Dr. Terry joined the A. V. M. A. in 1928. He was also a member of the Pennsylvania State Veterinary Medical Association.

HARRY W. PARMER

Dr. Harry W. Parmer, of Ide Grove, Iowa, died in a Sioux City, Iowa, hospital, December 11, 1929, after a brief illness. He was born at Minneapolis, Minn., March 17, 1893, and lived in that city until about ten years ago. He attended Iowa State College and received his degrees of B. S. and D. V. M., the latter in 1919. Dr. Parmer joined the A. V. M. A. in 1928. He was a member of the Iowa Veterinary Medical Association, the American Legion and the Masonic Lodge at Ida Grove. He is survived by his widow.

Dr. A. E. Howell, of Homer, Mich., died December 2, 1929, at the age of 92. He was said to be the oldest veterinarian in Michigan, having recently retired after sixty years of practice.

Our sympathy goes out to Dr. George A. Young, of Syracuse, Nebraska, in the death of his wife, November 30, 1929.

PERSONALS

MARRIAGES

Dr. Bernard Mann (U. P. '17) to Miss Helen Marie Marcus, both of Philadelphia, Pa., November 17, 1929, at Philadelphia.

Dr. B. W. Conrad (K. C. V. C. '07) to Mrs. Estella Masheter, both of Sabetha, Kansas, December 9, 1929.

BIRTHS

To Dr. and Mrs. Robert G. Little, of Williamsport, Pa., a son, Robert G., Jr., August 3, 1929.

To Dr. and Mrs. C. C. Middleton, of Birmingham, Ala., a daughter, Mary Ann, November 24, 1929.

To Dr. and Mrs. Harry A. Hoopes, of La Rue, Ohio, a daughter, Betty Frances, October 3, 1929.

To Dr. and Mrs. R. J. Rosselot, of Elyria, Ohio, a son, James Edward, October 7, 1929.

PERSONALS

Dr. E. H. Thornburg (Ind. '12) has resumed his practice in Lynn, Indiana.

Dr. E. C. Hughes (Ind. '16), of Carlinville, Ill., recently moved into new office quarters.

Dr. E. R. Carlson (Mich. '25) is studying human medicine at the University of Michigan, Ann Arbor.

Dr. J. V. Ramler (K. C. V. C. '18), formerly located at Wheaton, Minn., is now at Fergus Falls, Minn.

Dr. J. M. Tade (K. C. V. C. '03), of Vincennes, Ind., completed a new veterinary hospital, December 1.

Dr. H. R. Hornbaker (K. C. V. C. '13) has taken over the practice of Dr. O. T. Douglass at Galesburg, Ill.

Dr. W. R. Van Ness (Ont. '07-McK. '08) has requested a change of address from Lebanon, Ohio, to Mason, Ohio.

Dr. W. B. Coon (Ont. '12), of Forest Grove, Oregon, has a son (Elwyn W.) studying veterinary medicine at Iowa State College.

Drs. H. H. Rowe (Chi. '18) and Taylor P. Rowe (U. P. '18), of Richmond, Va., are now associated in practice at 316 N. Henry St.

Dr. J. J. Kavenek (T. H. '15), of Hartford, Conn., has been appointed Deputy commissioner on Domestic Animals of Connecticut.

Dr. O. T. Douglass (Iowa '27), who has been practicing in Galesburg, Ill., has removed to Davenport, Iowa, to engage in practice there.

Dr. Daniel De Camp (K. S. A. C. '29) has been transferred from Keokuk, Iowa, to the Swift & Company Produce Plant at Wichita, Kans.

Dr. J. D. Beck (U. P. '28) is instructor in veterinary medicine in the School of Veterinary Medicine, University of Pennsylvania, Philadelphia.

Dr. Thomas B. Carter (Ont. '14), formerly of Portland, Ore., is now located at Gresham, Ore., and is Dairy Herd Inspector for Multnomah County.

Dr. R. R. Dwyre (Ind. '23), of Oswego, Ill., was recently appointed Lee County (Ill.) Veterinarian. He will have his headquarters in Dixon, Ill.

Dr. H. T. Farmer (U. S. C. V. S. '11) is now located at 3100 North Ave., Richmond, Va., following the dissolution of the firm of Farmer and Rowe.

Dr. W. E. Welsh (Iowa '27), of Hibbing, Minn., has accepted a position in the Department of Veterinary Science, at the University of Wisconsin, Madison.

Dr. L. A. Mosher (Mich. '15), of the Fort Dodge Serum Company, has been transferred from Lima, Ohio, to Atlanta, Ga., c/o Southeastern Laboratories, Inc.

Dr. Andrew G. Vogt (Amer. '84), of Allenhurst, N. J., accompanied by Mrs. Vogt, is in Europe and will not return to the United States until June 1, 1930.

Dr. A. F. Nelson (Ind. '01-Chi. '02), of Thorntown, Ind., recently had one of his ankles badly sprained, when he was rolled on by a horse which he was treating.

Dr. C. L. Kern (Corn. '24), who is with the Dairymen's League Cooperative Association, has been transferred from Utica, N. Y., to Buffalo, N. Y. Address: 45 Allen St.

Dr. R. E. Nesbitt (Chi. '02), who has been Logan County (Ill.) Veterinarian for the past two years, has returned to Clinton, Ill., and resumed his general practice there.

Dr. E. L. Sidwell (K. C. V. C. '15), of Hardin, Ill., recently fractured two ribs, when he fell from a ladder while attempting to swing a crippled horse in the barn of one of his clients.

Dr. B. A. Utter (McK. '16), formerly of Welcome, Minn., is in charge of poultry inspection with the Bureau of Agricultural Economics at the Hormel Packing Company plant, Austin, Minn.

Dr. G. R. Deeren (Chi. '15), who has been in the employ of the Illinois State Department of Agriculture, recently opened an office in Taylorville, Ill., where he will engage in general practice.

Dr. C. D. Evans (McK. '16), of Kane, Pa., has assumed charge of the certified milk plant of the F. M. Johnson and Son Ideal Farm, of Kane, Pa. Dr. Evans continues to conduct his general practice.

Dr. C. E. Swink (Iowa '27) is now Dairy and Food Inspector of Hibbing, Minn., having resigned his position at Austin, Minn., on poultry inspection work with the Bureau of Agricultural Economics.

Dr. J. T. Redmon (Ind. '09), of Covington, Ind., took the Pasteur treatment, during the early part of December, following a bite on one of his hands inflicted by a horse that exhibited symptoms of rabies.

Dr. Robert Learmonth (Mich. '25), who has been conducting a small-animal practice in Denver, Colo., for several years, has accepted a position at the Rhode Island Agricultural Experiment Station, Kingston.

Dr. G. E. McIntosh (U. S. C. V. S. '23), of Louisville, Ky., is now connected with the 3-D Thoroughbred Breeding Farm, at Arlington, Texas, and will remain in the Lone Star State until about April 1, 1930.

Dr. Carl E. Chase (O. S. U. '28), who has been associated with Dr. F. F. Russell (Ont. '13), of Concord, N. H., for some time, has opened an office for general practice in Manchester, N. H. Address: 72 Blodget St.

Dr. Arthur Trickett (K. C. V. C. '01), of Kansas City, Mo., was official veterinarian for the American Royal Live Stock and Horse Show held in Kansas City, November 16-23, 1929. Dr. Trickett has held this honored position for several years.

Dr. W. N. Armstrong (Ont. '94), of Concord, Mich., was presented with a beautiful traveling bag by the Jackson County Board of Supervisors recently, as a token of the Board's appreciation of the work Dr. Armstrong has done for his county during the past few years.

Dr. J. J. McDowell (Ont. '11), of Britton, S. Dak., wrecked his car near Barry, Minn., while returning from Minneapolis, the first week in December. A cow crossed the road directly in front of his car and Dr. McDowell took to the ditch, to avoid hitting the cow. No one in the car was injured.

Dr. Edw. Lapple (Cin. '11), of Fostoria, Ohio, was among the out-of-state veterinarians who attended the three-day veterinary short course held at Purdue University, November 12-13-14, 1929. Dr. Lapple remained over for an extra day, to take some special work in which he was interested.

Dr. M. C. Hawn (Iowa '27), formerly of the veterinary staff of the University of Wisconsin, has accepted a position in the Veterinary Department of the North Dakota Agricultural College, instead of joining the staff of the North Shore Animal Hospital, as announced in the November issue of the JOURNAL.

Dr. Frederick P. Ruhl (Amer. '85), of Milford, Del., started on a trip December 20 which will take him to Havana, Cuba; Kingston, Jamaica; Nassau, Bahamas; and the Panama Canal. Considering the fact that Dr. Ruhl has been in general practice continuously since 1885, a vacation is very much in order at this time.

Dr. R. E. Rebrassier (O. S. U. '14), of the Department of Pathology, College of Veterinary Medicine, Ohio State University, has been appointed to the staff of the Ohio Experiment Station and will conduct work at the State Serum Institute, at Reynoldsburg, Ohio. Dr. Rebrassier will divide his time between the University and the Experiment Station.

Dr. A. W. Heflin (St. Jos. '19), of Gower, Mo., drove his brown mare, Madge Kennedy, in the class for roadsters, hitched single to bikes, at the horse show held in connection with the American Royal Live Stock Show, at Kansas City, November 21, 1929. At one of the turns, Dr. Heflin fell and was dragged about 25 yards, but escaped without serious injuries.

Dr. S. E. Hershey (Queen's U. '98), of Charleston, W. Va., has been making investigations in connection with several outbreaks of the pulmonary form of hemorrhagic septicemia among cattle, at the request of Hon. John L. Smith, Commissioner of Agriculture. Dr. Hershey reports the losses from the disease this year as being considerably less than they were one year ago.

Dr. R. P. Dingman (Ont. '27), of Prophetstown, Ill., has been having unusually good success with his show pigeons this fall. At the Mid-South Fair, held in Memphis, Tenn., in October, he showed twenty-six birds and captured two first prizes, two second, one third, three fourth and three fifth. The following week, he showed them at the Arkansas State Fair and won seven first prizes, four second, one third, one fourth, one fifth and one sixth.

Dr. Geo. H. Glover (Iowa '85), dean of the Veterinary Department, Colorado Agricultural College, received the largest number of votes to determine the citizen who had done the most for Fort Collins (Colo.) during the year 1929. The members of three civic clubs, Kiwanis, Lions and Rotary, balloted to decide who should receive the honor, symbolized by a loving cup donated by Mr. and Mrs. Beverly Irwin and presented to Dr. Glover at the annual meeting of the Fort Collins Chamber of Commerce, December 9.

Dr. C. M. McFarland (K. C. V. C. '00), who has been associated with the Sihler Serum Company as Secretary-Treasurer and Sales Manager for the past nine years, resigned recently to become associated with the Allied Laboratories with headquarters in Chicago. Dr. McFarland will be Sales Manager of Serum and Virus. Before going with the Sihler Serum Company, Dr. McFarland was in the U. S. Bureau of Animal Industry and at various times was stated at St. Joseph, Mo.; Spokane, Wash.; and Sioux City, Iowa.